THE American JOURNAL OF Surgery

FEBRUARY 1956

THURSTON SCOTT WELTON, M.D., EDITOR

Parotidectomy for Mixed Tumors

Multiple Polyposis of Small Intestine

Benign and Malignant Tumors of Ureter

SIXTY-FIFTH YEAR OF PUBLICATION

FEBRUARY 1956

THE American JOURNAL OF Surgery

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(1) Farmer, D. A.: M. Clin. North America *38*:1403, 1954. (2) Lichtman, S. S.: Diseases of the Liver, Gallbladder and Bile Ducts, ed. 3, Philadelphia, Lea & Febiger, 1953, vol. 2, pp. 1146, 1150, 1302. (3) Rosser, J. H., and McCall, R. A.: U. S. Armed Forces M. J. *5*:658, 1954. (4) Schwimmer, D.; Boyd, L. J., and Rubin, S. H.: Bull. New York M. Coll. *16*:102, 1953. (5) Best, R. R.; Rasmussen, J. A., and Wilson, C. E.: A.M.A. Arch. Surg. *67*:839, 1953. (6) Pearse, H. E.: Am. J. Surg. *89*:1191, 1955.

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1. Eckenhoff, J. E.; and Dripps, R.D.: Anesthesiology, 15:681, Nov., 1954.

2. Sokoloff, Louis; King, B. D.; and Wechsler, R. L.: Med. Clin. North America, 38:499, Mar., 1954.

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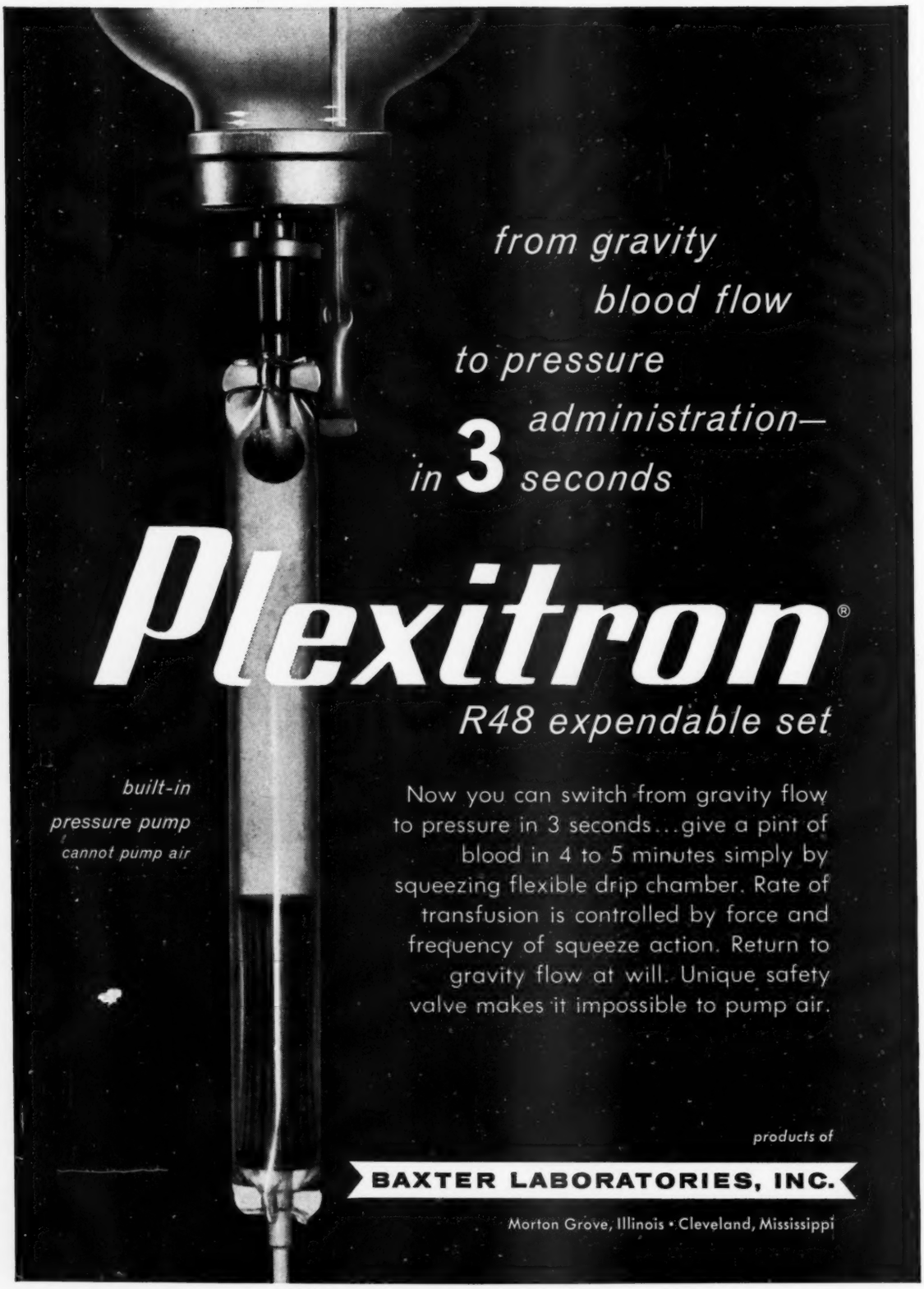
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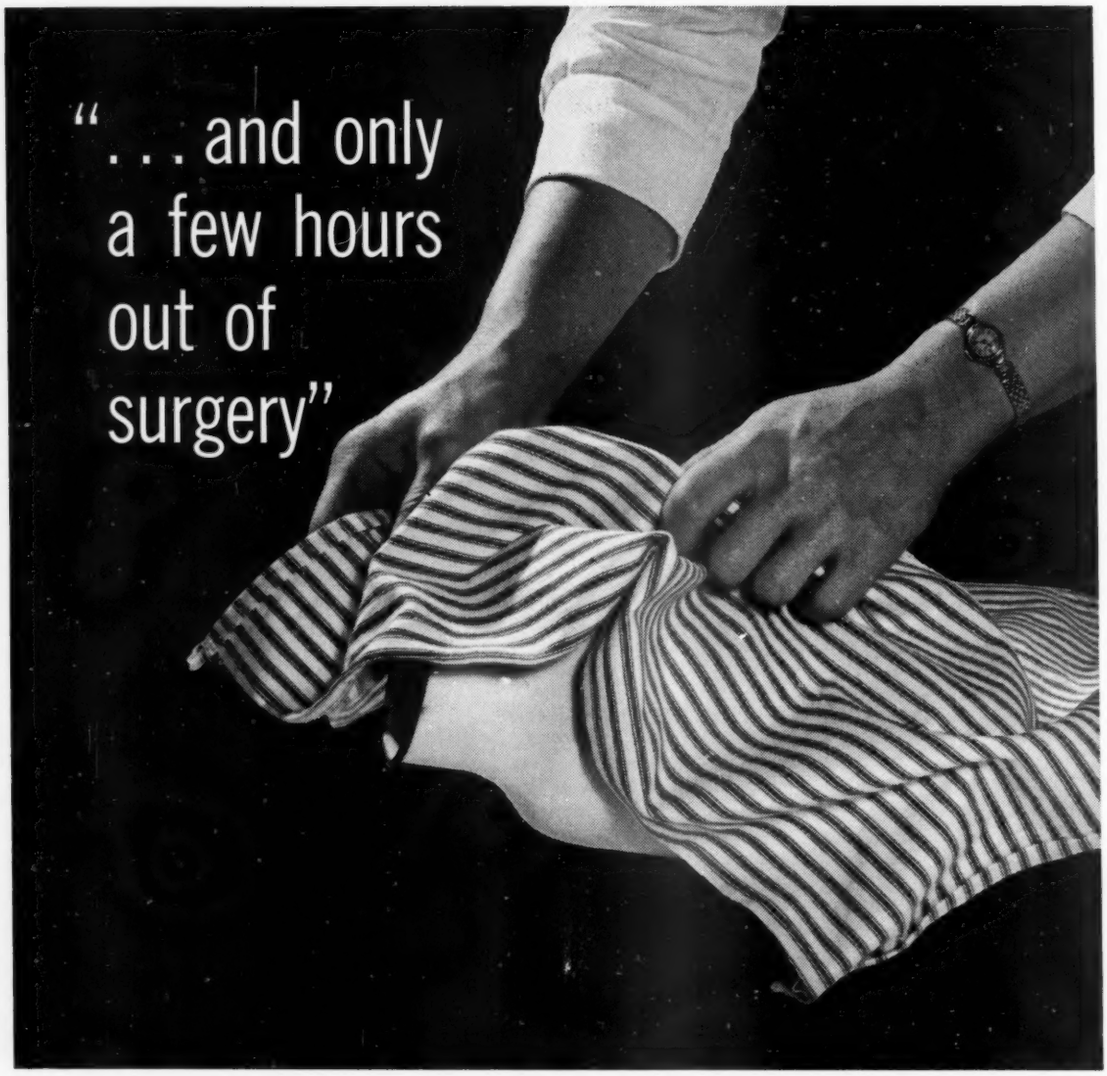

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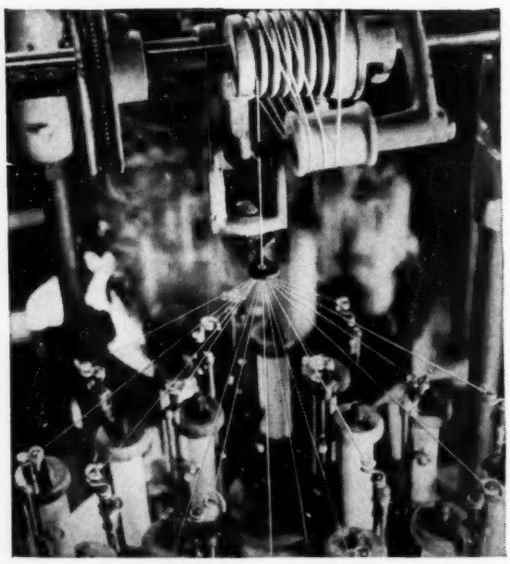

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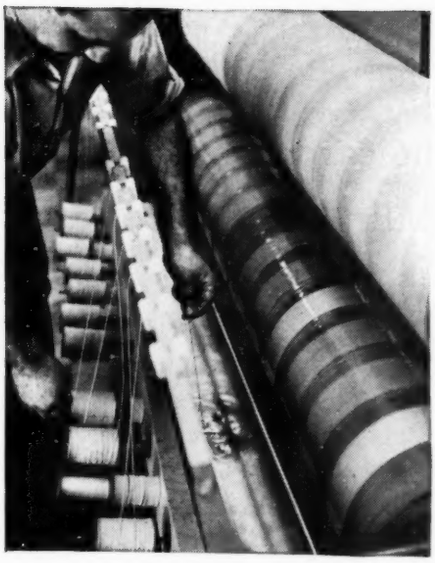

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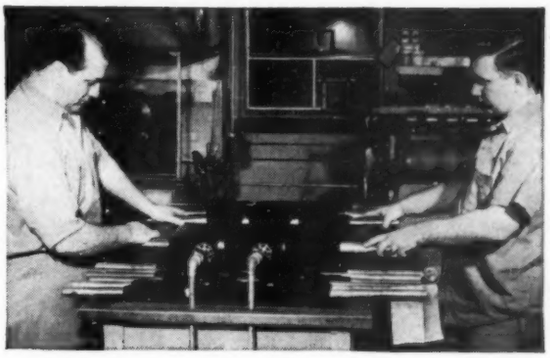

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It was further found that various combinations of adrenochrome, notably the oxime and semicarbazone, produced stable solutions. But, these were so slightly soluble that sufficient concentration could not be obtained for practical therapeutic use. By combining these adrenochrome compounds in a sodium salicylate complex a stable, soluble form can be obtained. This complex has been given the generic name, carbazochrome salicylate, and is supplied under the trade name Adrenosem Salicylate.

Roskam, in his study entitled "The Arrest of Bleeding,"[2] enumerates "the drugs whose efficaciousness as hemostatics have been proved by accurate methods in experimental animals and in healthy men as well. . . . One is the monosemicarbazone of adrenochrome [Adrenosem Salicylate]."

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(* U.S. Patent 2,581,850)

Sherber, in an early study,[3] concludes that Adrenosem Salicylate* "is a potent antihemorrhagic factor in those conditions in which the integrity of the smaller vessels is interrupted, and is superior to any similar material that is now available."

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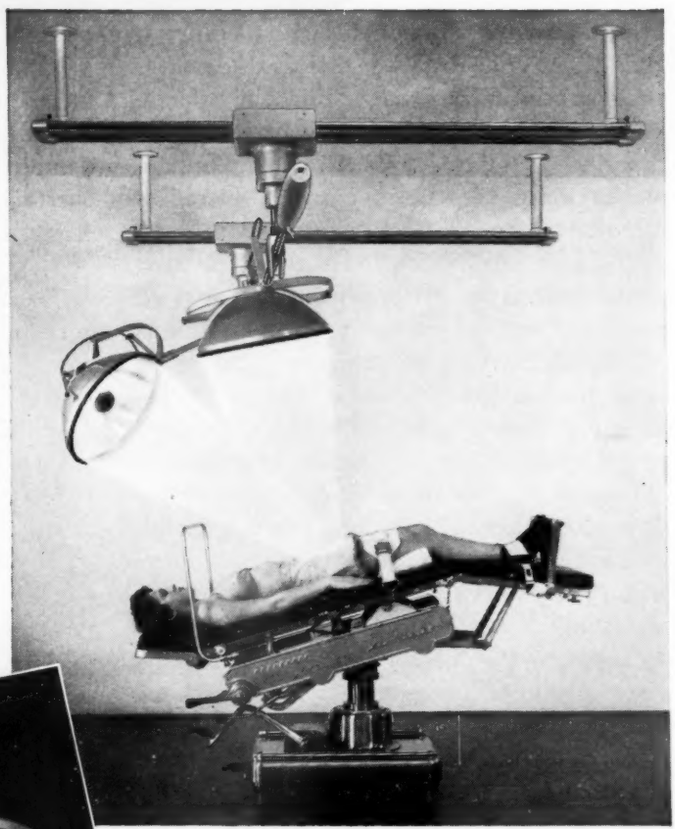

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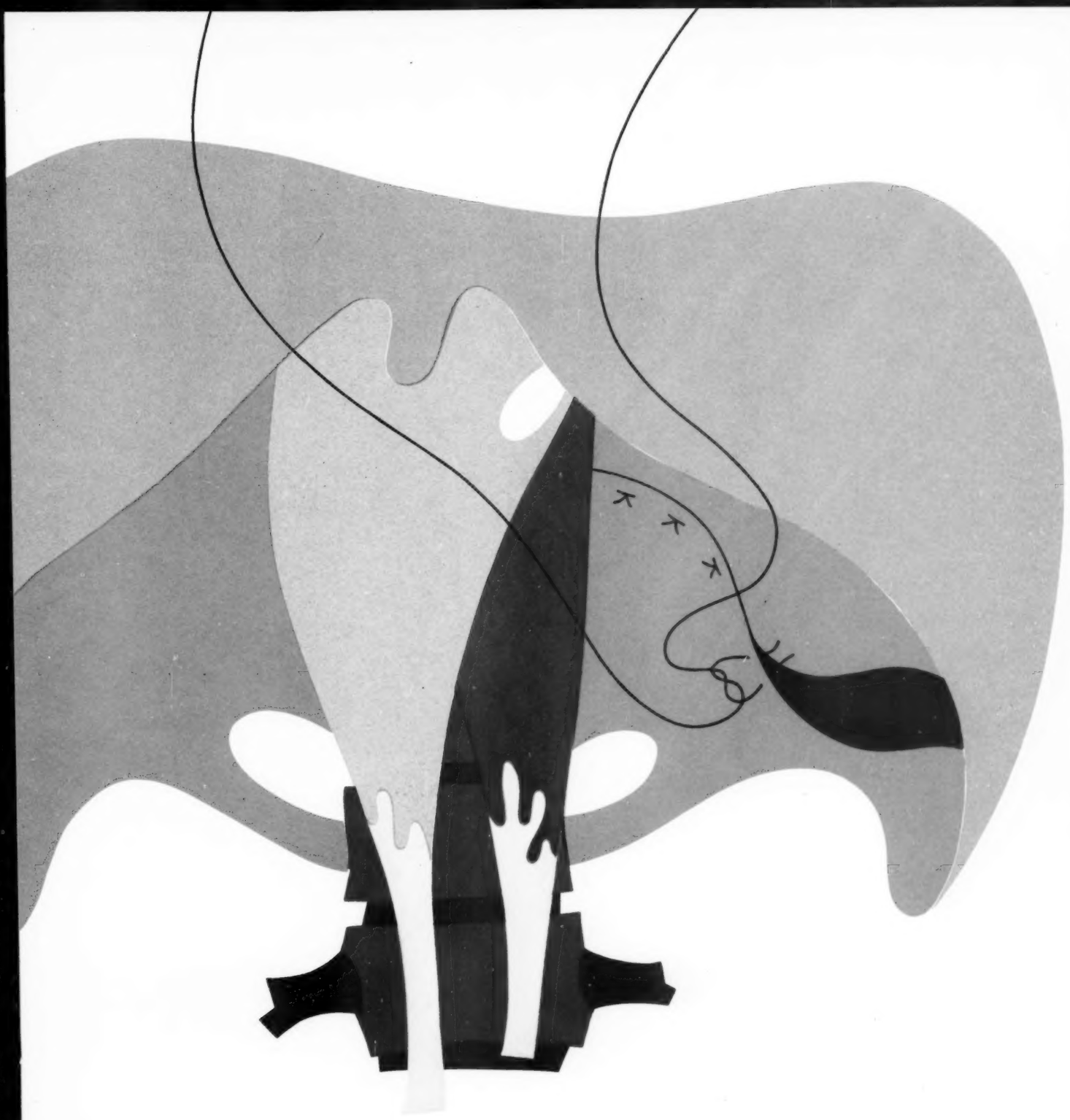

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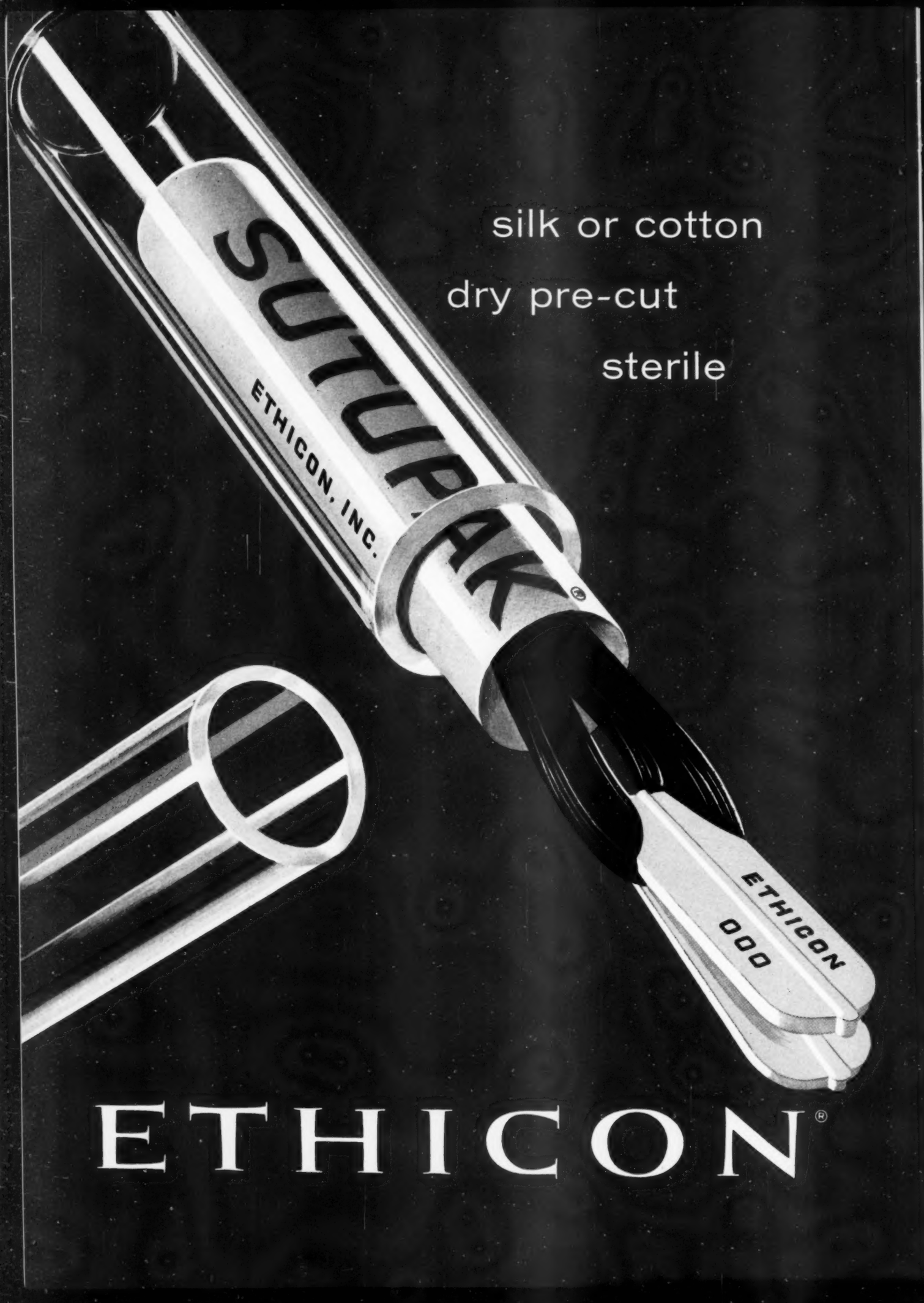
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1. Overall, J. C.: Southern M. J. 47:789, 1954. 2. Editorial: New England J. M. 246:111, 1952.
3. Grayzel, H. G., Heimer, C. B., and Grayzel R. W.: New York St. J. M. 53:2233, 1953.
4. Heimer, C. B., Grayzel, H. G., and Kramer, B.: Archives of Pediatrics 68:382, 1951.
5. Behrman, H. T., Combes, F. C., Bobroff, A., and Leviticus, R.: Ind. Med. & Surg. 18:512, 1949.
6. Turell, R.: New York St. J. M. 50:2282, 1950.

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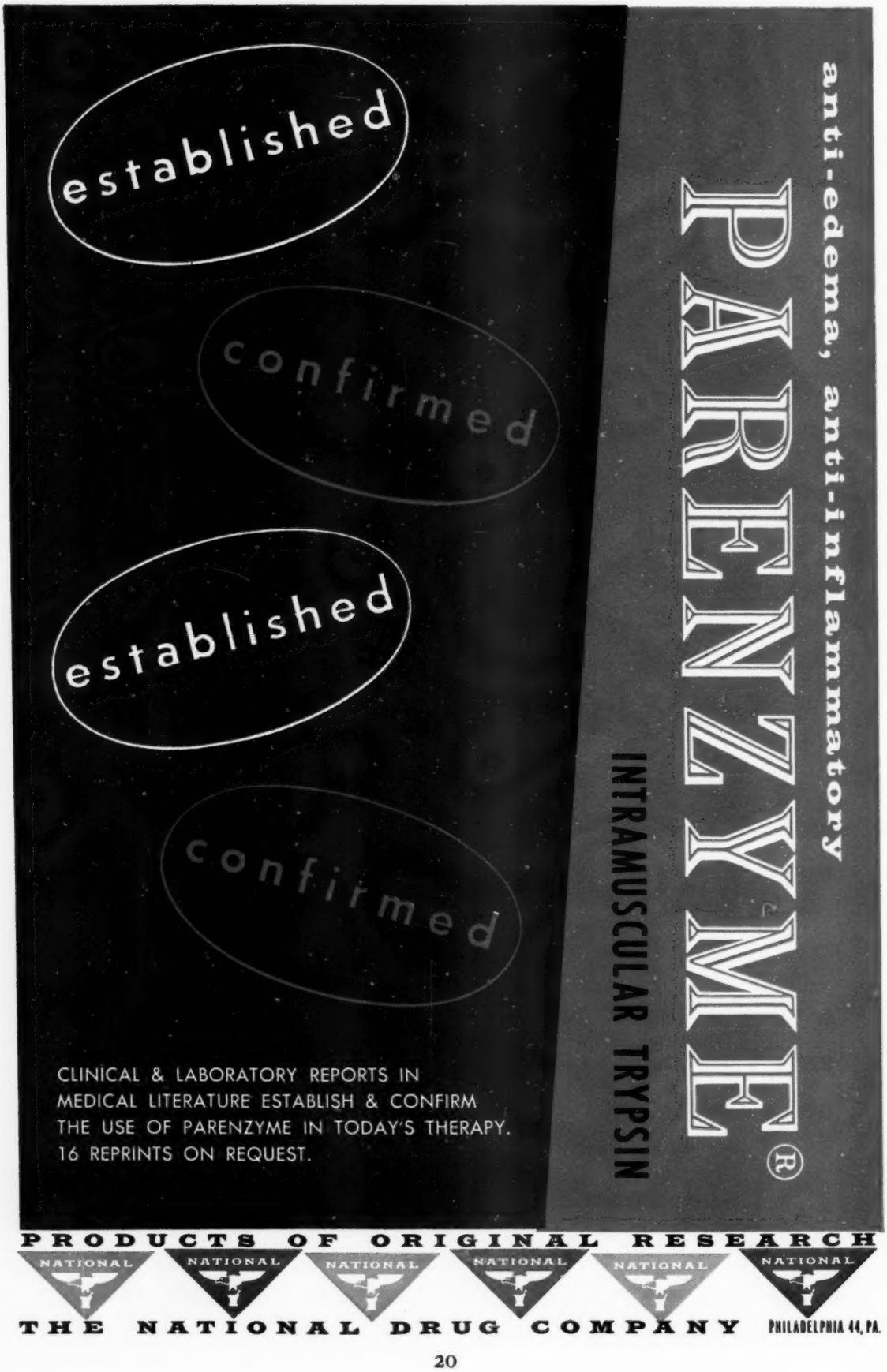

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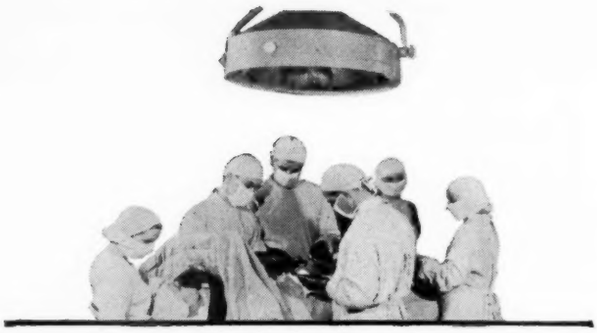

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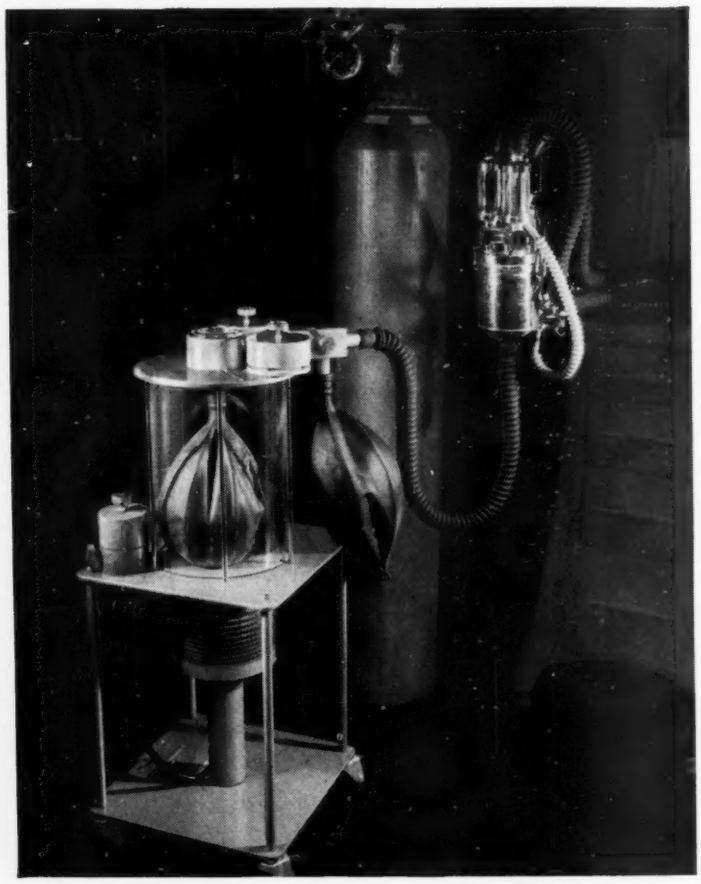

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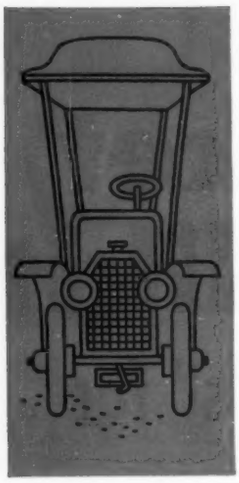

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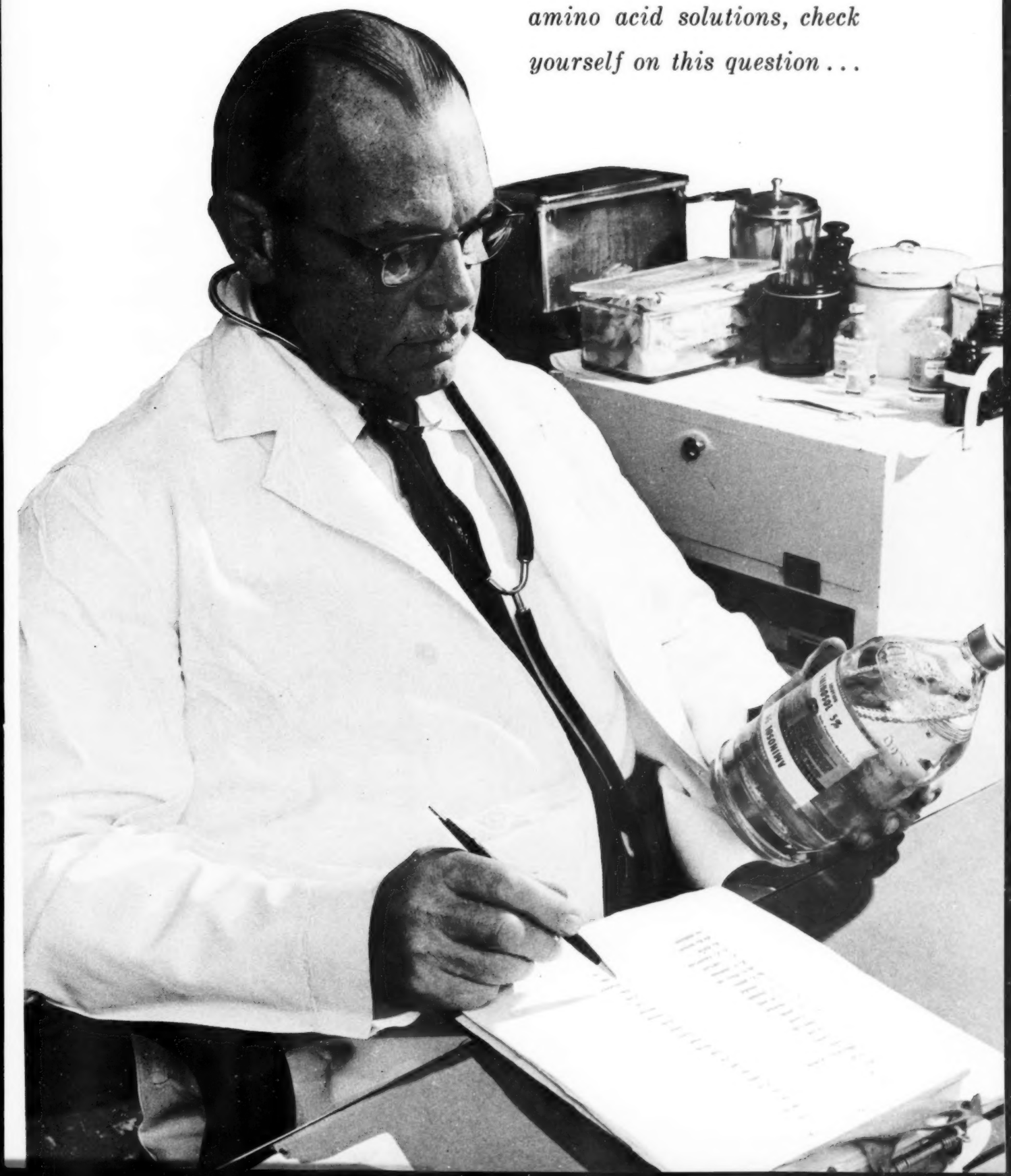

Why does one protein hydrolysate bring about more rapid recovery than another?

If protein hydrolysates differ in nutritive quality—and they do—the place to start looking is in the protein sources themselves. You'll need to know what amino acids are present—and in what proportion. (A variation in content of any one of the essential amino acids makes quite a difference, clinically.)

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Cannon et al.[1] – in a 10-day feeding of protein-starved rats—found that AMINOSOL produced approximately 50% more weight gains than either an acid hydrolysate of casein or a beef plasma hydrolysate. Frost et al.[2] rank it considerably above casein hydrolysate for plasma regeneration and nitrogen repletion. Carroll[3] notes that AMINOSOL is "well tolerated and efficient when given intravenously . . . When studied by other workers, it has proved to be of the highest nutritive value."

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1. Cannon, P. R., Frazier, L. E., and Hughes, R. H. (1950), Protein Hydrolysates—Food for the Sick, Research Reviews, Office of Naval Research, November, p. 13. 2. Frost, D. V., Heinsen, J., and Olsen, R. T. (1946), Intravenous Use of High Levels of Fibrin and Casein Hydrolysates in Hypoproteinemic Dogs, Arch. Biochem., 10:215, June. 3. Carroll, W. W. (1954), Parenteral Fluids in Gastrointestinal Surgery, Surgical Clinics of No. America, Vol. 34, No. 1, Chicago Number, February.

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References: 1. N.N.R. 1954, p. 107. 2. J.A.M.A. 153: 1516 (Dec. 26) 1953.

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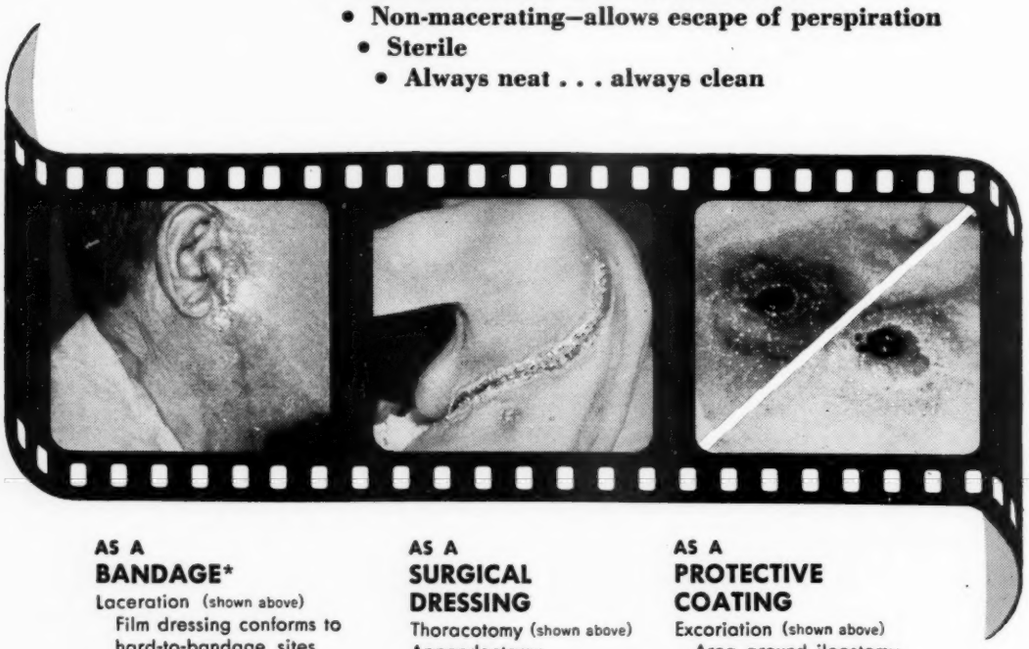

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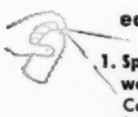

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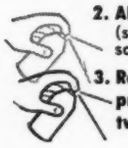

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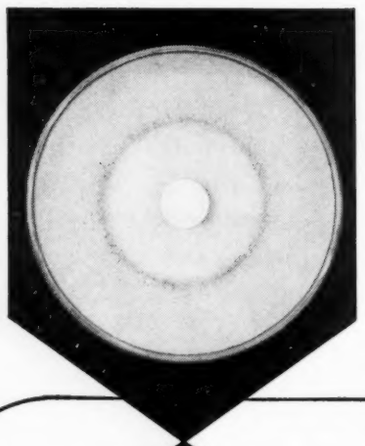

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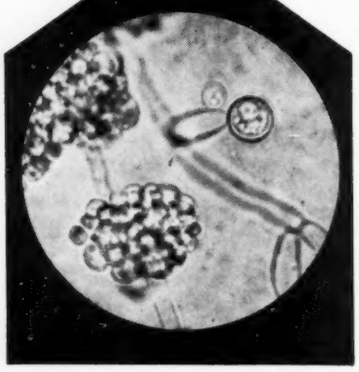

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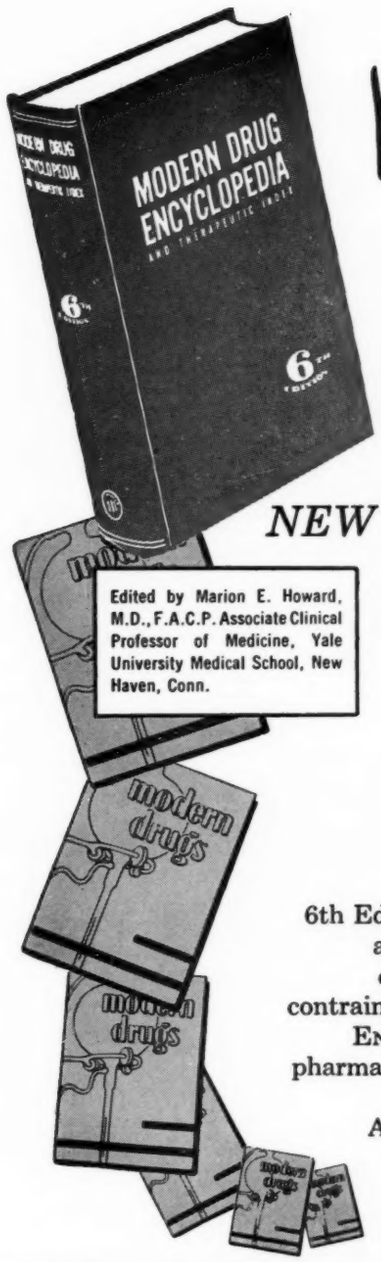

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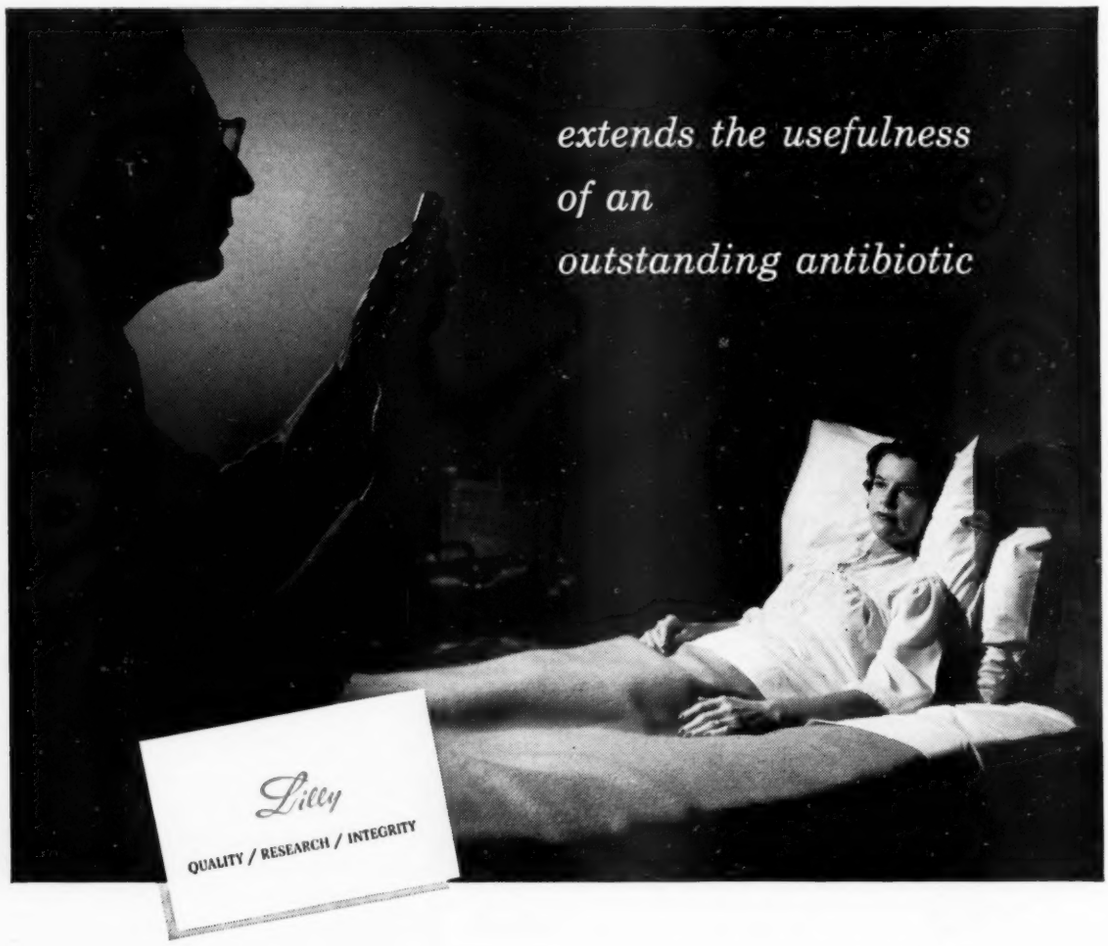

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Editorial

PSYCHOLOGIC ASPECTS OF DISTURBED NEUROMUSCULAR FUNCTIONING IN THE PARAPLEGIC*

PARAPLEGIA is a rather unique field of medicine. Other specialties are limited more or less to one aspect—the patient may have a heart condition, tuberculosis or a dermatologic problem. The traumatic spinal cord lesion has not only neurologic problems but also many other organic complications secondary to paraplegia. In order to understand some of the emotional conflicts that may arise in connection with the paralysis, some of the difficulties such a patient must undergo on his road to "rehabilitation" will be reviewed.

Following injury the average patient knows only that his "back is broken." He soon learns from his physician that some tests must be performed in order to ascertain whether an operation on his spinal cord is necessary. The physician studies the spinal dynamics and informs the patient that "a block exists." This means very little to the patient in most instances, even after a thorough explanation. He is told that laminectomy should be performed in the hope that his limbs might move voluntarily again.[1] Each postoperative day he tries to "wiggle a toe," move his foot, or stroke his skin for "feeling"—but to no avail. It is at this time that the patient usually realizes he is not going to walk again, especially after the decompressive operation of the spinal cord does not give the "hoped for" miraculous results. The earlier laminectomy is performed, the better is the chance of effecting some return of function.[1] However, if the spinal cord tissue has been destroyed, there is no functionally valuable regeneration. Return of any of the sensory modalities or motor power indicates that there was edema rather than interruption of the spinal cord. This holds true whether or not laminectomy has been performed. The so-called "walking paraplegic" usually has a cauda equina lesion. Infrequently, "walking paraplegics" can be found among those who have incomplete spinal cord lesions.

Following World War II many paralyzed patients felt strong resentment against the government, holding it responsible for their injuries. Postwar patients who sustained the injury in an automobile accident were more apt to blame themselves and become depressed.

Three to six months after injury, whether or not laminectomy has been performed, the average patient turns his thoughts once more to the problem of living. The patient who formerly was a "driver" becomes one again; the boy who wanted to become an attorney arranges to attend law school. However, the patient who was maladjusted before injury is a postinjury problem. The alcoholic continues to drink; the inadequate, shiftless individual has difficulty with his job training. The shy, retiring person becomes more withdrawn and should be contacted early by the psychologists in order to halt further withdrawal.

When the neurologic problems are solved, the patients must face the inevitable complications of paraplegia.[2] One of the major complications

[1] COMARR, A. E. and KAUFMAN, A. A. A survey of the neurological results of 858 spinal cord injuries. *J. Neurosurg.* To be published.

[2] BORS, E. V. A. Technical Bulletin 10–503. Washington, D. C., Dec. 10, 1948.

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is decubitus or pressure ulcers.[3] While the patients are still confined to bed, decubiti frequently develop over the bony prominences such as the trochanters, sacrum, malleoli and heels. Later, when the patients are able to be up in wheel chairs, decubiti form over the ischial tuberosities. These usually require surgery with its attendant emotional upsets.

In at least 52 per cent (complete lesions) to 72 per cent (incomplete lesions) of patients with spinal cord injuries reflex bladders or bladders that are amenable to extrinsic pressure develop spontaneously.[4] The remaining patients must have transurethral vesical neck resections or various nerve operations, or both.[5,6] During this period many sieges of pyelonephritis with chills and fever may develop. Perhaps a penoscrotal fistula will develop as a complication of the intraurethral catheter.[7] This may require three or four operative procedures until satisfactory bladder function is attained. The patient must wear a "catcher" for the urine and must constantly be on guard when in public.

A patient with a complete lesion has no voluntary control of his rectal sphincters. He attempts to retrain his bowel movements by means of daily trips to the bathroom where a combination of suppositories and enemas or digital stimulation, or both, are used. Such procedures may have an adverse effect on the patient's esthetic sense, and frequently are the sources of inferiority feelings.

If the patient has an upper motor neuron lesion, the chances are good that spasticity will develop.[8] A slight to moderate amount is useful, but often the spasticity becomes severe enough to cause the knees to rest nearly on the chest, or to be so continuously clonic that sleep and rest are virtually impossible. Emotional trauma apparently aggravates spasticity.[9] Clinically, it is apparent that spasticity is increased by a variety of stimuli such as kidney or bladder stones, urinary tract infections, a full bladder from a "plugged" catheter, a bedsore, or even tight-fitting shoes. Pinching or stroking the skin will bring forth spasticity in the patient with an upper motor neuron lesion.

Pain is another complication of marked magnitude found usually in patients with cauda equina lesions. There have been numerous instances when a patient's emotional tensions have played an important part in his attitudes toward pain. In such cases the psychologists have been of invaluable assistance. All patients who are being considered for neurosurgical procedures for the alleviation of pain are routinely seen by the psychologist and psychiatrist. Their evaluation and recommendations are important in the final decision. It should be noted that to date spinothalamic chordotomy is successful for the "shooting" type of pain but not for the "burning" type of pain.

Many of the aforementioned problems might be tolerated by the paralyzed patient if he were fortunate enough to live a normal sex life, but often this also has been affected. The incomplete lesions retain sexual abilities in accordance with the amount of damage to the nerves involved in sexual activities. My observations reveal that the majority of patients with complete upper motor neuron lesions can have erections by stimulation of the organ. The majority cannot ejaculate. Many of these individuals can have cohabitation with gratification for the mate, but they themselves do not experience orgasm. The majority of patients with complete lower motor neuron lesions have neither erections nor ejaculations. However, I have seen two patients with complete lesions at thoracic four level who have fathered children. Findings from seminal tract washings,[10] when compared with normal values, indicated that artificial insemination by this method offered a poor chance of impregnating the mate. However, if a donor's specimen is mixed with

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[10] Comarr, A. E. and Bors, E. Spermatocystography in patients with spinal cord injuries. *J. Urol.*, 73: 172, 1955.

that of a patient, he may believe it was his spermatozoa which fertilized his mate.

The relationship between degree of incapacity and personal and social adjustment of patients with cord injury has been described by Seymour.[11] In contrast to the average patient, who is hospitalized for a short time and thanks the physician profusely when he is discharged, is the paraplegic and quadriplegic who generally finds this difficult to do. The hours, days and sometimes years spent in preventing or correcting the multitude of complications would, thus, not appear to be universally appreciated. The reason for this is not a genuine lack of gratitude but rather the fact that these patients came into the hospital paralyzed and are wheeled out paralyzed, in spite of their hope to leave under voluntary locomotion.

The physician and psychologist meet the challenge of these difficult conditions with their attendant complications and emotional problems, and together they return a good percentage of remarkably adjusted and well rehabilitated patients to the community. It is only after a thorough understanding and knowledge of what the patient's burden is that such a resynthesis may be achieved.

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SCIENTIFIC PAPERS

Treatment of Multiple Polyposis of the Small Intestine

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ABOUT once in a lifetime an active surgeon might expect to encounter polyposis (or adenomatosis) of the small intestine as the cause of intussusception, or as the source of gastrointestinal bleeding of a subacute or chronic nature. The problems of diagnosis and treatment are not those of congenital colonic polyposis, or of isolated gastric or small intestinal polypi. The purpose of this paper is to review some of these problems and suggest a method of therapy not previously described.

History. Although there are observations in the literature concerning this disease, for example, those of Hutchinson in 1896 and Weber in 1919, it remained for Peutz[1] to describe the disease entity in 1921, after reviewing the literature. Jeghers[8] more recently brought the disease into focus and detailed its exact hereditary nature.

Incidence. Since Jeghers' analysis of the disease, case reports have increased in number until they are now widespread among journals in almost every clinical field of medicine.[4–9,13,15] This increasing interest will, I predict, remove polyposis from the category of rare diseases.

The frequency with which malignant changes occur in multiple small intestinal polyposis is not clear. Neither is it clear just how many of the more than 350 cases[15] of intestinal polyposis were or could have been multiple polyposis of the small intestine. The incidence of malignancy is recorded as 24 per cent,[15] but one gains the impression that death from anemia and multiple intussusception in the absence of a definitive attack on the disease itself prevents many of these patients from reaching the age of maximum incidence of cancer.

Forms of the Disease. This disease entity consists primarily of hundreds of adenomatous polyps of the small intestine, although there are sometimes associated polyps of the stomach or colon, or both, usually fewer in number. There are some who look upon this disease as polyposis of the entire intestinal tract. Peutz's description of pharyngeal polyps lends some credence to this belief, but the predominant view is that the polyps are always in the small intestines and may or may not be elsewhere. The polyps vary in size from 1 mm. to 3 cm. Usually the larger polyps have long thin stalks, traction on which accounts for the tendency to intussusception. There are two forms of the disease: a true hereditary type, and a sporadic or non-hereditary type. The hereditary type, sometimes referred to as the Peutz-Jeghers syndrome, has been described as a conjugate of melanin spots (Figs. 1 and 2) and small intestinal polyposis. This is explained by assuming that a single pleiotropic gene is a simple dominant. The melanin spots are usually striking in their concentration and may be located around the mouth, or on the lips, palate, labia, anus or digits. The spots vary in size from 0.5 to 5 mm., and from brown to blue-black in color. This, combined with a family history of multiple polyposis, makes the diagnosis possible during routine physical examination or examination during other illnesses. The sporadic or non-hereditary group may or may not have melanin spots to attract the examiner's attention, and are therefore most apt to be met during a search for the cause of secondary anemia, subacute gastrointestinal hemorrhage, or as an acute surgical emergency due to intussusception.

1 2

FIG. 1. Melanin spots on lips and circumoral. Note variation in size.

FIG. 2. Melanin spots on fingers. Note how inconspicuous spots are in both Figures 1 and 2. (Case from University of Michigan Hospital. Courtesy of Drs. A. Bortz and F. Bethell.)

FIG. 3. Intussusception and small intestinal polyps. Upper small intestine by double contrast technic.

Diagnosis. In general, it may be said that the clinical symptoms recorded in most of the case reports are intermittent epigastric distress, secondary anemia and an occasional diarrheic bowel movement, with or without melena. There is some reason to believe that the epigastric distress is due to a recurring and remitting partial intussusception that only occasionally becomes complete. When melanin spots are found the diagnosis is established; and if there is also a positive family history, the diagnosis is doubly confirmed.

In the acute phase intussusception becomes irrevocably established and produces complete obstruction. Occasionally scout films will show intussusception as the cause of intestinal obstruction preoperatively. Since single small intestinal polyps are commonly the cause of

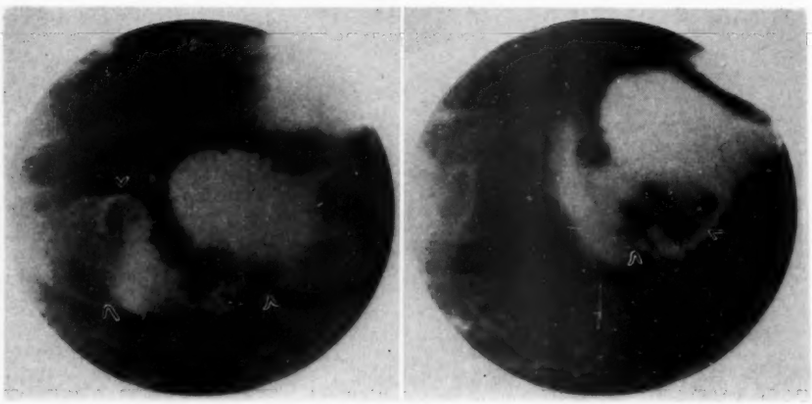

FIG. 4. Detail showing polyps. (Case from Ohio State University Hospital. Courtesy of Drs. E. Ellison and J. Morton.)

intussusception, one must bear in mind the possibility of multiple polyposis so that it may be confirmed or disproved at operation for the intussusception. One of our patients had been operated upon three times for intussusception before the true nature of the disease was suspected. Melanin spots were not seen in this patient until a fifth operation was performed. Rarely, if ever, will small intestinal multiple polyposis be the cause of massive gastrointestinal hemorrhage sufficient to create a surgical emergency.

In the subacute or chronic phase, that is, in cases of unexplained melena with secondary anemia and the vague epigastric distress of recurring and remitting intussusception, x-ray studies will rarely be of direct assistance, except to find the rarer case of associated gastric or colonic polyposis, or both. While such findings in a patient with melanin spots and a family history of polyposis support the diagnosis, the absence of spots or a family history, or both will usually be misleading. In subacute or chronic intestinal bleeding in patients with unexplained vague abdominal distress, roentgen examination of the entire gastrointestinal tract seems mandatory before operation, or after operation to supplement the operative findings. Special emphasis should be placed on small intestine double-contrast technics, since even careful palpation at operation does not always disclose the number or extent of the polyps. Figures 3 and 4 illustrate a rare exception in which x-rays clearly demonstrated both the polyps and intussusception. This patient, who was observed at the University Hospital of Ohio State University, is a unique example. As this disease becomes better known it should be recognized clinically early in its course. The description of the melanin spots in connection with polyposis has already alerted the internist, pediatrician and dermatologist, and the new double-contrast x-ray technic for examining the small intestine will confirm the diagnosis with increasing frequency in the future. In those patients without melanin spots and a family history the condition will continue to be found by the surgeon. One of the purposes of this paper is to remind the surgeon, internist, pediatrician and dermatologist to consider this disease in making a diagnosis, and to plan a future elective attack on the polyps directly.

Treatment. Treatment in the past has consisted of meeting the emergency by excision of the intussusceptum of the small bowel, discovering and treating any associated colonic and gastric polyps, combating the anemia by means of conservative management, and making frequent observations for possible malignant degeneration. The aforementioned patient, who had undergone three operations for intussusceptions, required a fourth operation for melena and secondary anemia. During this operation (performed in August, 1953) a method of direct attack on the polyps in the small intestine and the colon was discovered. A thorough search of the literature failed to show a report of any similar technic. Further experience has added minor variations, uncovered some of the method's limitations, and extended its usefulness to other conditions of the bowel lumen. A description of the procedure follows.

After thorough bowel preparation, a 4 cm.

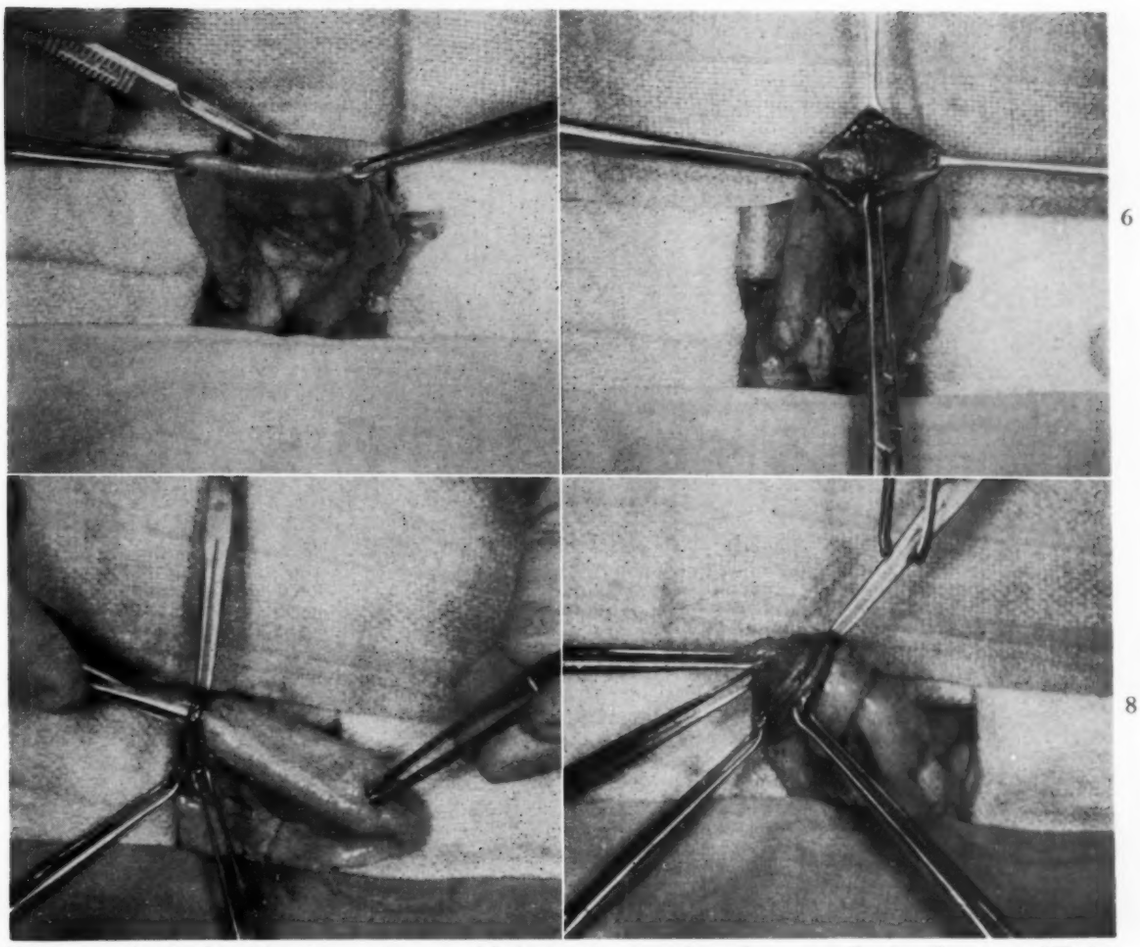

FIG. 5. Longitudinal incision (measuring 4 cm.) being made approximately 25 cm. distal to ligament of Treitz.

FIG. 6. The lumen is held open by four Allis forceps (low tension or "soft").

FIG. 7. A long (8 inch) Allis forceps (soft or low tension) has been thrust into lumen and full-thickness antimesenteric bowel indented between jaws by blunt thumb forceps. This is usually inserted a full 8 inches instead of the 4 inches shown here.

FIG. 8. Slow and gentle traction on the inserted Allis forceps with countertraction on the four Allis forceps on the edges of the incision has begun to extrovert the jejunum.

longitudinal incision is made on the antimesenteric side of the intestine approximately 25 cm. distal to the ligament of Treitz. (Fig. 5.) Four soft Allis forceps hold this incision apart (Fig. 6), while a long (8 inch) soft Allis forceps is thrust as far as possible up the lumen and the jaws opened against the antimesenteric side. With the tip of the finger of the other hand or with a closed hemostat or thumb forceps, full thickness of the bowel is indented between the jaws of the forceps, and the jaws are closed on full-thickness intestine. (Fig. 7.) Traction on this forceps, with countertraction on the four instruments on the edge of the enterotomy incision, begins to invert or intussuscept this segment into the lumen. (Fig. 8.) It is often helpful to exert a little pressure from the outside by the same finger tip or blunt instrument that was used to engage the full thickness of intestine in the forceps within the lumen. Great care must be exercised not to tear the intestine, and the key to this is to allow enough time for the intestine to stretch and mold with steady but not excessive traction. Occasionally the proximal portion nearest the enterotomy incision will evert first instead of the distant portion, and this variant is useful when one does not know how far a fat mesentery will

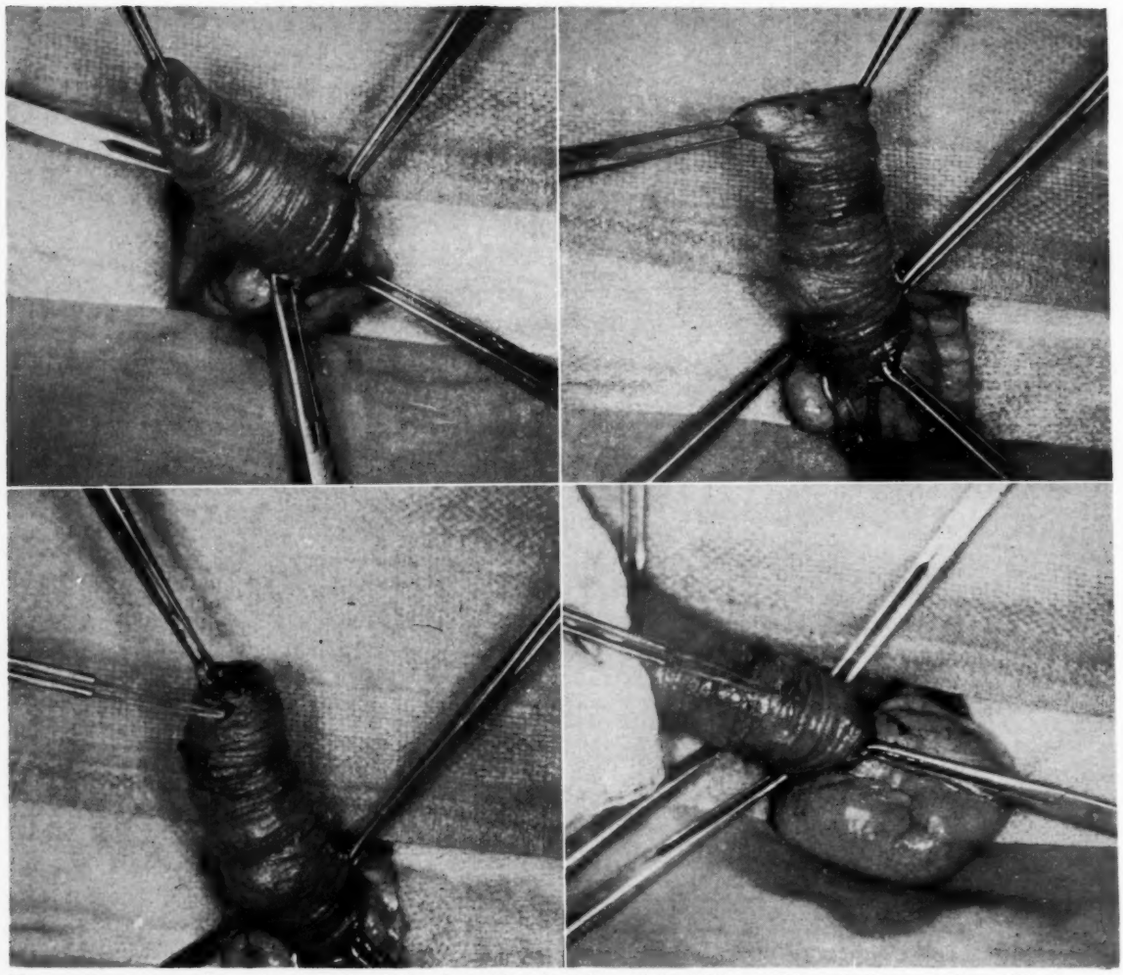

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FIG. 9. Further prolapse has occurred slowly, still using the original bite and just before another Allis forceps is inserted to increase the extroversion. Note that no tearing has occurred.

FIG. 10. Prolapse is two-thirds complete and a "hand over hand" method is in progress with the Allis forceps in the lumen. Two small polyps may possibly be seen but are not typical since these photographs were taken at the fifth operation (the first operation after the original cautery polypectomy eighteen months before).

FIG. 11. The prolapse is now complete. Note gathering of intestine. One polyp is shown about to be cauterized. Hundreds of polyps existed here at the fourth operation (first operation at which this procedure was used).

FIG. 12. Another small polyp is being cauterized. The intestine is slightly cyanotic at the enterotomy incision and this slow change occurs usually when the limits of prolapse are reached. There is also a rather abrupt resistance to further traction so that it is not difficult to know when to stop.

restrict the length of segment that can be exposed. In this instance a hand over hand encouragement of this prolapse with Allis forceps on the folds of the mucosal lumen is indicated. Some rocking and the employment of additional forceps as the process of extroversion proceeds turns the small intestine inside out to full view (Figs. 9 and 10) for inspection, biopsy and removal of the polyps by snare, cautery, knife or scissors. (Fig. 11.) The maximum length of intestine thus exposable seems to be about 20 to 25 cm. As the limit of extroversion is approached (Fig. 12), the prolapsed portion will become cyanotic and will fill and stretch the incision. One must be careful not to exceed this point, which is usually rather definite in both the feel and appearance; otherwise ecchymoses, hematomas and tearing will result. When the intestine is replaced, the portion of the bowel distally may be prolapsed through the same incision in a like manner, thus covering a maximum of almost 3 feet for each

enterotomy incision. Before the distal portion is replaced, Allis forceps should be applied intraluminally, as at the start of the extroversion, to mark the limits of the inspected and treated intestine. In this way, as the bowel is replaced a black silk suture may be taken through the serosa to mark the limit externally. Since this is usually greater than the length of the forceps, one may temporarily mark the external location of this point by externally applied forceps as the intestine is replaced, before releasing the inside (long) Allis forceps. Some such method of marking must be used if the lumen of the entire small intestine is to be inspected and treated. The enterotomy wounds are closed transversely in two layers. (Fig. 13.)

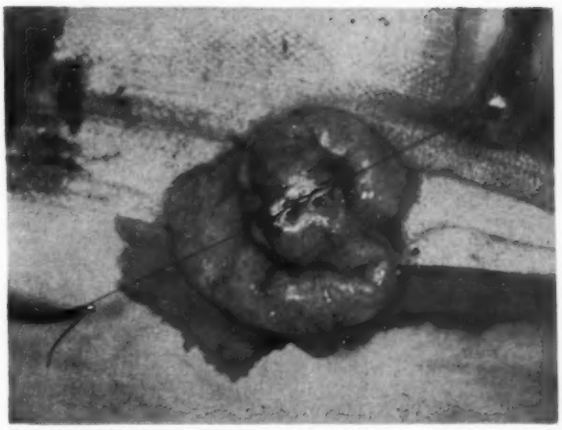

FIG. 13. The prolapse has been reduced, the distal portion extroverted and replaced, and the original longitudinal enterotomy incision closed transversely in two layers. A silk suture (not shown) has been taken in the serosa of the distal limit of exposure to allow proper measuring to the next distal enterotomy incision.

The postoperative course of patients so treated has been smooth. In one case follow-up reoperation eighteen months later for leiomyoma of the uterus showed no adhesions except of the omentum to the abdominal wall and a residual of two 4 mm. polyps. (Fig. 11.) During the interval without treatment the patient had remained well, with no anemia. The operation previously described was employed on portions of the colon in this patient and in one other. The degree of laxity of the mesocolon and the amount of fat in the omentum or epiploica causes greater limitations in the amount of prolapsible colon than in the small intestine. We have not yet performed this operation on the stomach, but in the colon it has proved very useful in the search for polyps and ulcers, and in inspecting the proximal and distal segments for implants or secondary growths in determining whether the limits of resection for carcinoma have been adequate. Whether any relaxant drugs such as levophed® would be of value I do not know, but this will be the subject of further investigation, possibly to increase the extent of each exposure and decrease the number of separate enterotomies required.

SUMMARY

The problems of diagnosis and treatment of multiple polyposis of the small intestine are different from those of congenital colonic polyposis, or of isolated gastric polypi or small intestinal polypi. A feature of the hereditary type of multiple polyposis is the finding of melanin spots on the body; this may or may not occur in sporadic or non-hereditary polyposis. Symptoms include occasional diarrhea with or without melena, secondary anemia, and epigastric distress probably due to incomplete recurring intussusception. Physicians are urged to consider this disease when making a diagnosis, so that an elective attack may be made on the polyps directly. Thus, in many cases death from anemia and multiple intussusception can be prevented.

A practical method has been devised for exposing the mucosa of the small intestine for excision or destruction of the polyps, or both. The method is also useful, to a limited extent, in examining the colon for polyps, ulcers and cancer implants. Inspection of portions of the colon and small bowel may help in determining the adequacy of mucosal resection for carcinoma or ulcers, or both.

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We recommend:

Essentials of Orthopaedics. By Philip Wiles, M.S. (Lond.). Second edition, 538 pages, 393 text figures with 7 color plates. (Corrected and revised, with ten articles on new material.) Boston, 1955. Little, Brown & Co. *Price* $10.00.

The Spine. A Radiological Text and Atlas. By Bernard S. Epstein, M.D. 539 pages with 721 illustrations on 331 figures, 37 page bibliography. Philadelphia, 1955. Lea & Febiger. *Price* $16.50.

Total Parotidectomy for Mixed Tumor

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MIXED tumors of the parotid are the most common neoplasms of salivary gland origin. Although they are not malignant by usual criteria,[1] neither are they strictly benign. It is possible that they represent, as suggested by Willis,[2] a stage in progression between a benign and a definitely malignant lesion.

That these tumors are likely to recur is well recorded, such recurrence depending on the type of treatment and length of time the patients are followed up. Statistics on recurrence vary but a conservative estimate of the average rate is about 25 per cent.[3]

A number of factors contribute to this high figure: (1) One cause is incomplete removal due to reluctance on the part of the surgeon to carry out a wide excision in an area traversed by the facial nerve. Lack of understanding of the anatomy of the area and unfamiliarity with the problem result in timid excision, too frequently done with local anesthesia under the impression that these tumors are always benign. (Fig. 1.) (2) There can be error in diagnosis. I have seen a number of patients with recurrent tumors who were first operated upon under local anesthesia for "biopsy of a lymph node" or "removal of a cyst," resulting in an incomplete removal of a parotid tumor after a tedious and distressing episode for both patient and physician. (3) The presence of satellite nodules, a characteristic of these multicentric tumors, is another factor producing recurrence when the tumors are removed by the usual extracapsular dissection which may allow daughter nodules to remain. This has been mentioned by McFarland,[3,4] who found that small tumors tended to recur more frequently than large tumors. He speculated that surgery may best be delayed to allow coalescence of these nodules, i.e., that the tumor be allowed to "ripen" before excision. (4) Careful microscopic study of mixed tumors will show penetration of the capsule by tumor cells (28 per cent in some series)[5]—another invitation to recurrence.

Because of this real threat of recurrence, the increased risk of facial nerve injury with subsequent operations in a scarred area and the possibility of malignant change[6] or primary low grade malignancy, it is evident that the fate of the patient with a mixed tumor depends on the surgeon who operates upon him for the first time. Therefore, the first operation must be thorough, to reduce recurrence, and at the same time should avoid injury to the facial nerve.

FIG. 1. Recurrent mixed tumor two years after incomplete removal through inadequate, poorly placed incision.

Diagnosis. The diagnosis of parotid tumor is usually not difficult although a firm cyst deep in the gland may accurately simulate neoplasm. I have been impressed by the frequency of lesions occurring in the isthmus of the gland (nine of twenty in the series reported herein) where they may be mistaken for enlarged lymph nodes.

The differential diagnosis between carcinoma and benign tumor is possible clinically in most instances. Important features are listed in Table 1. Of particular significance is the presence of pain, facial paralysis or metastases. Some malignancies tend to be flat, infiltrative

TABLE 1

Features	Benign or Mixed Tumor	Malignant
Duration	Long	Short
Age (yr.)	40−	40+
Size (variable)	Slow growth	Rapid or increasing
Contour	Projects	May be flat, infiltrative
Consistency	Firm-hard	Hard
Mobility	Slight	More fixed
Pain	Not usual	Frequent
Paralysis	Unusual	Frequent
Metastases	None	Frequent

and less conspicuous, and do not displace the lobe of ear as do the benign tumors. I have not taken preliminary biopsy specimens because of danger of implanting tumor cells, but I have exposed the tumor at operation and have taken biopsy specimens only of those suspected of being malignant. The difficulties in interpreting frozen sections of these tumors make this of value only in unequivocal carcinomas. I have not found sialography to be of value in differentiating tumors, but it will demonstrate obstruction to the ducts.

Clinical error occurred in three "mixed tumors" situated deep in the gland. On removal by total parotidectomy they were found to be densely encapsulated cysts. It may be that a biopsy specimen should be taken at operation in each case to prevent this occurrence, but mixed tumors may undergo secondary necrosis.

Anatomic Considerations. Probably the first accurate description of the surgical anatomy of the parotid was given by Bailey[7] who confirmed the earlier findings of McWhorter,[8] demonstrating that the facial nerve does not pass through the gland in a strict sense but is situated between the superficial and deep lobes, enclosed in the parotidomasseteric fascia. Bailey advocated removal of the entire gland with preservation of the nerve, and he presented a scheme for mobilizing the superficial lobe. Further detailed description of the facial nerve has been given by McCormack et al.[9]

Technic. Since 1946 I have employed total parotidectomy in the treatment of mixed tumors and during this period have performed the procedure twenty-three times, an additional patient having been treated by superficial lobectomy. The operation is performed under general intratracheal anesthesia the patient being draped with the ear exposed for orientation. A "J" or "Y" incision[10,11] is made and the superficial lobe of the gland exposed. Dissection is then begun along the anterior margin of the superficial lobe, identifying the branches of the facial nerve and tracing them in a centripetal direction. To facilitate this the parotid duct is identified, divided and the proximal end is grasped with a forcep and, using this as an instrument of traction, the superficial lobe is gently elevated. Once the two major divisions of the nerve are identified (as they fork about the isthmus) the dissection of the isthmus and deep lobe is not difficult, provided it is not hurried and is done carefully. The normal relationship of the nerve may be distorted by tumor and the operator must be alert to this. The peripheral portions of the nerve are not usually thus displaced.

I have not found it necessary to ligate the external carotid, but the careful maintenance of hemostasis is essential. In one patient the main trunk of the facial nerve was identified first and dissection carried peripherally from this point, but the approach seemed more difficult. In only one patient with a superficial tumor was superficial lobectomy done, preserving the deep lobe. Superficial lobectomy does not appear to be a wise procedure since the difficult part of the dissection is completed when the superficial lobe is mobilized and the nerve exposed.

MATERIAL

A summary of the patients treated is presented in Table II. It will be noted that of the twenty patients with primary mixed tumors, simple excision was performed in two. One patient was so selected because of age (sixty-one) and three previous cerebrovascular accidents. Local anesthesia was used but is not recommended for the usual case. In the other patient the tumor was pedunculated and attached to the parotid by a thin stalk. Total

TABLE II

Type of Tumor	No. of Cases	Location		Injury to Nerve		Gustatory Sweating	Total Parotidectomy	Superficial Lobectomy	Excision
		Superficial	Deep or in Isthmus	Temporary Weakness	Weakness, Portion of Mandibular Division				
Primary mixed tumor	20	11	9	7	1	2	17	1	2
Recurrent mixed tumor	2	1	1	..	1	..	2	..	..
Adenolymphoma	1	1	..	..	..	1	1	..	..
Cyst	3	2	1	1	..	..	3	..	..

parotidectomy had been contemplated, but the findings at operation suggested that simple excision including the parotid attachment might be sufficient.

One patient with an adenolymphoma was subjected to total parotidectomy. Clinically this was thought to be a mixed tumor and a biopsy specimen was not taken at operation. Adenolymphomas are uncommon tumors, probably are entirely benign, but may recur if incompletely removed. It is likely that extracapsular excision of this tumor would have been adequate treatment and that taking a biopsy specimen at operation would have revealed its true nature. Differentiation of an adenolymphoma from a mixed tumor is not possible on clinical grounds; to date there is no record of one being diagnosed preoperatively.

As indicated previously, total parotidectomy was performed on three patients for cysts within the gland. In each instance the cyst was densely encapsulated and was thought to be a mixed tumor. Histologic examination, however, failed to reveal tumor in the wall of the removed specimens and the etiology of these is obscure, there being no evidence of duct obstruction. Again, taking a biopsy specimen at operation may have identified these lesions as cysts, but an occasional mixed tumor may undergo cystic degeneration or inflammatory change to cause further confusion.

RESULTS

Recurrences. There have been no recurrences in this series—the average length of follow-up is thirty-three months with extremes of one month and eighty months. Admittedly, this is too short a period to be significant since McFarland has recorded recurrences (new tumors?) forty-five years after removal,[3] but most recurrences will appear within four years and the majority of these occur in the first year.[6] However, if one is permitted to compare these with mixed tumors of the submaxillary which rarely recur after total submaxillary excision,[12,13] a prediction can be made that total parotidectomy may produce similar results. Also, daughter tumors and extracapsular penetrations will be removed by total parotidectomy, whereas they may be allowed to remain when extracapsular dissection is done. (Figs. 2 and 3.) The difficulty of excising the tumor and a rim of normal parotid to encompass these nodules is obvious.

Facial Nerve Injury. Permanent complete facial nerve paralysis has not occurred, but two patients have slight non-disfiguring weakness of a portion of the mandibular division; one of these patients was operated upon for recurrent tumor. Eight additional patients have had temporary weakness of one or more of the divisions of the nerve (usually mandibular). This usually appears on the day following surgery, suggesting edema and functional interruption of the nerve, and has cleared completely in several weeks except for the two patients with persistent weakness. Treatment of this condition has been support to the angle of the mouth by tape or an acrylic loop and galvanic stimulation to preserve muscle tone pending recovery of function.

Three patients have had sweating of the cheek on the operated side associated with meals ("gustatory sweating," Frey syndrome).

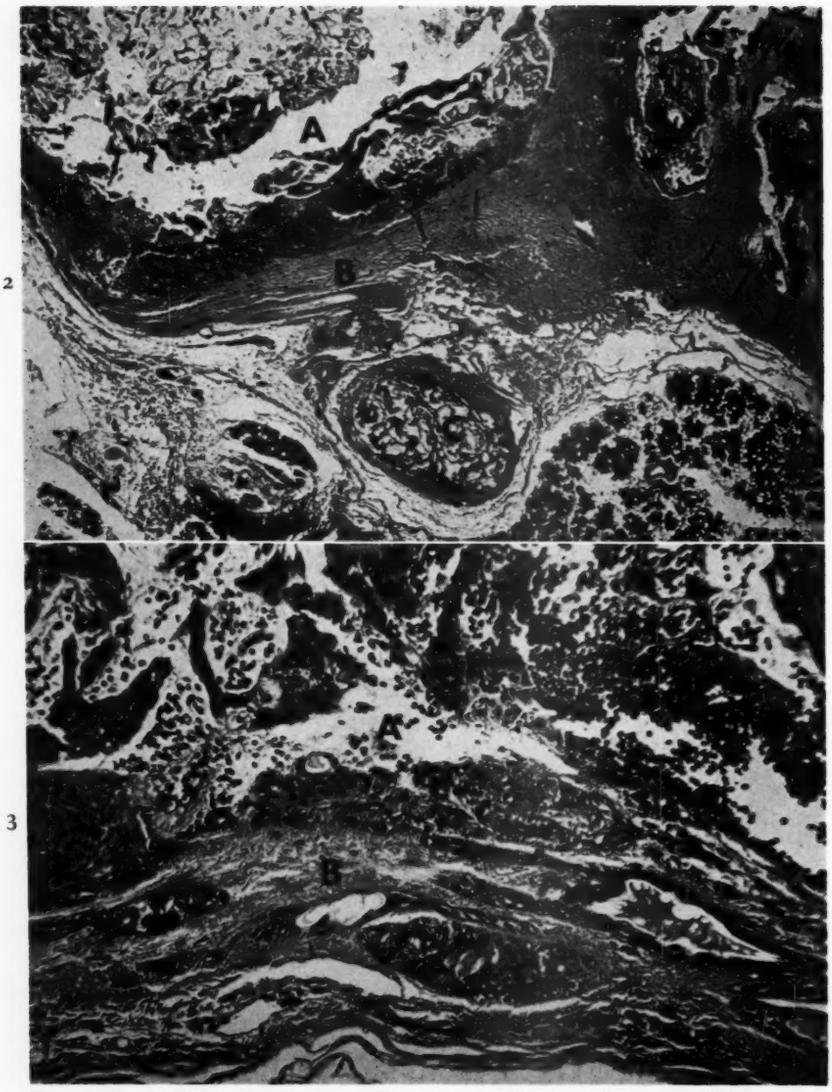

FIG. 2. Typical daughter tumor (C) separated from main tumor (A) by capsule (B). Extracapsular removal of tumor may allow satellite nodule to remain. (Original magnification, × 15.)

FIG. 3. Penetration of capsule (B) by tumor cells (C). (Original magnification, × 100.)

This has not been of major consequence and has been alluded to by others.[14]

Cosmetic result has been excellent, the incision being carefully placed in a skin fold. The depression in the cheek following removal of the gland fills in and leaves little if any facial asymmetry.

CONCLUSIONS

1. Total parotidectomy with preservation of the facial nerve has been performed on twenty-three patients.

2. Prolonged follow-up is necessary to evaluate statistically this procedure, but it should yield improved results for stated reasons.

3. The procedure is technically feasible but should not be undertaken without full understanding of the anatomy involved and should be done only under general anesthesia.

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Textbook of Endocrinology. Edited by Robert H. Williams, M.D. Contributors: Wm. H. Daughaday, Peter H. Forsham, Harry B. Friedgood, John Eager Howard, Edward C. Reifenstein, Wm. W. Scott, George Van S. Smith, George W. Thorn, Lawson Wilkins, Robert H. Williams. Second edition, 172 figures. (Worthy of the highest praise.) Philadelphia, 1955. W. B. Saunders Co. *Price* $13.00.

Cancer Cells. By E. V. Cowdry. 677 pages, illustrated, 63 page bibliography. (Written by a master scientist.) Philadelphia, 1955. W. B. Saunders Co. *Price* $16.00.

Detoxification and Augmented Anesthesia of Procaine by Thiamin (Vitamin B_1)

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FIFTY years ago Alfred Einhorn first synthesized procaine. It has been widely used for many years for local, regional and spinal anesthesia. More recently it has been used intravenously and orally for general analgesia, cardiac irregularities, disorders of the gastrointestinal tract, and for an increasing variety of other conditions. Comparatively speaking, it is the safest and most widely used anesthetic drug available, yet at times its toxicity may become manifest.

This paper deals with some experimental and clinical observations to show that the toxicity of procaine is decreased by thiamin chloride, and that the anesthetic action is at the same time augmented. This information should be welcomed not only by surgeons and anesthetists but also by internists.

The remote, although nevertheless possible neurologic complications, respiratory embarrassment and even death from spinal anesthesia (even when procine is used), are fully appreciated by anesthetists.[1–7] Certain pathologic conditions and narcotics have been found to reduce the lethal dose of procaine intracisternally.[8,9] With the proper selection of the patient, correct technic of administration and proper dosage, these complications should rarely be encountered. They are more apt to occur when the person giving the anesthesia is inexperienced, when errors in technic or dosage are accidentally made, or when the patient may have an idiosyncrasy or allergy to the drug.

The occasional late complications of spinal anesthesia such as diplopia, atrophy or paralysis of isolated muscles, and psychic reflex sequelae, appear to be unrelated to the type of drug used. These are more apt to be due to such intrinsic and hidden factors in the patient as might be connected with unrecognized disease, particularly one involving the central nervous system. Unrecognized vitamin deficiencies, particularly of vitamin B_1 or B complex, may be contributing causes for such complications.

For obvious reasons death from spinal anesthesia probably occurs more often than has been recorded in the literature. The more potent anesthetic drugs are the cause more often than procaine, yet it has happened and can happen with the latter. One of the recognized causes of death from spinal anesthesia is the direct paralyzing or toxic effect of the drug on the vital centers in the medulla oblongata, particularly the respiratory centers. This has been demonstrated experimentally.[9] While some may say this does not happen clinically,[10] in any event respiration usually stops for some minutes before the heart stops beating. This unfortunate event may follow improper positioning of the patient after the introduction of the spinal anesthetic and from failure to appreciate the hydrodynamics of the introduced anesthetic solution in the column of spinal fluid. This is apt to happen particularly in short and stocky individuals. Usually acute bulbar anemia, shock or vasomotor paralysis is given as the cause of death from such respiratory failure.

It has been shown, although its significance is not fully appreciated, that the base of an anesthetic drug is more readily liberated in an alkaline solution, thus increasing the anesthetic efficiency.[11] Cancer patients and cachectic individuals may have an alkaline spinal fluid with a pH as high as 8.6.[12] Hence, in patients with an alkaline spinal fluid there may be an intensification of the anesthesia, and this in itself may account for some of the complications of spinal anesthesia. It is a simple matter to determine the actual pH of the spinal fluid at the time of anesthesia by the use of pH papers or an electric potentiometer.

The presence of an acid, on the other hand, inhibits anesthesia.[13] Therefore, it would be preferable to choose an anesthetic drug with a low pH to obviate the possible undesirable

rapid liberation of the anesthetic base by an alkaline spinal fluid.

Thiamin chloride (vitamin B_1) is an acid salt. Given alone intraspinally it specifically stimulates respiration and the sympathetic nervous system when introduced into the cisterna magna of cats.[14] No other drugs or chemicals are known to have this same direct stimulating action on respiration. Nicotinic acid, nicotinamide, hydrochloric acid (1:1000), ephedrine sulphate, adrenalin,® ergotamine tartrate, caffeine sodium benzoate, strychnine, camphor in oil, atropine, pituitary extracts, ammonium chloride and histamine diphosphate (1:1000) all failed to stimulate respiration when given by this route. The component parts of thiamin, thiazole and pyrimadine likewise failed to have the same stimulating effect as the whole vitamin. It would appear then that the stimulation by this vitamin is not due to irritation or change in the pH of the cerebrospinal fluid in the immediate vicinity of the vital areas, but that it is probably due to the unique oxidative and catalytic properties of this vitamin on the nerve cells in these centers.

In an emergency there are obvious advantages in the use of a prompt and effective drug such as vitamin B_1 intracisternally to restore respiration over the intravenous administration of the usual respiratory stimulants, especially when circulation is also impaired. Artificial respiration should always be maintained until one is sure the respiratory centers have returned to their complete automatic activity, and then for some time thereafter to be sure the centers will not again cease to function. If artificial respiration is stopped too soon after restoration of spontaneous breathing, respiration may cease again, but this time perhaps beyond recovery.

That vitamin B_1 alone can be safely given clinically via lumbar intraspinal injection is attested to by the hundreds of cases of various diseases of the nervous system in which this treatment was used.[15–31]

Experimentally, intracisternal injection of vitamin B_1 in cats appears to offer the vital centers some protection against illuminating gas poisoning, electric shock, cobra venom (neurotoxin) poisoning, and deuterium oxide (heavy water) poisoning.[12] There are no reports in which the vitamin has been given clinically intracisternally to overcome complete paralysis of respiration in such emergencies or in bulbar poliomyelitis, but it is worth while keeping in mind. It is estimated that 50 mg. of pure thiamin chloride would be an effective dose intracisternally for this purpose in adults. This vitamin should not be used if it is known that it enhances the toxicity of an anesthetic drug responsible for the respiratory paralysis (*vide infra*). It should be emphasized here that in the course of the experiments to be described it was found that when the respiratory centers are paralyzed by an anesthetic like procaine for more than eleven minutes, there is practically no hope for the vitamin to re-establish spontaneous breathing. The cardiac centers are less readily affected by the anesthetic; and unless the respiratory centers are made to react quickly within this grace period, the cardiac centers cease to function soon thereafter and the animal dies.

There has been only one clinical report of untoward effects from the intraspinal administration of vitamin B_1.[32] These effects may have been due to the use of a vitamin B_1 preparation containing a preservative such as chlorbutanol which is a fat solvent and detrimental to nerve tissue.

Thiamin chloride lowers the pH of procaine hydrochloride when mixed in solution and lowers its oxidation-reduction potential as tested with an electric potentiometer. This may explain in part why the *toxicity* of procaine is lowered by this vitamin.

DESCRIPTION OF EXPERIMENTS

To determine whether vitamin B_1 would be of value in conjunction with the use of anesthetic drugs, a series of 273 adult cats and five Rhesus monkeys were given intracisternal injections under light ether anesthesia. The introduction of an anesthetic drug or combination of drugs into the cisterna magna is the most accurate way of determining the toxic dose, as evidenced by respiratory paralysis. Subarachnoid hemorrhage or puncture of the cord by the needle vitiates the test. The former may so dilute the subsequent injection as to make the observations valueless. The latter usually results in prompt death of the animal or may cause ataxia or paralysis, usually of the hind limbs. Only those animals were considered in which the cisternal tap afforded a free flow of clear or only very slightly blood-tinged spinal fluid, and when one could be sure that no injury to nerve tissue by the needle had occurred.

Of the 273 cats, thirty-two were discarded because of these technical difficulties. With increased experience, only one cat was discarded in the last 100 used. Two hundred seventy-four injections were given into the cisterna magna of 241 cats, and the observations on these animals follows.

The immediate effects of various concentrations and amounts of procaine hydrochloride, thiamin hydrochloride and mixtures of these two drugs upon the vital centers were observed. A comparative study of a number of spinal anesthetic drugs was likewise made. An attempt was also made to determine the extent and duration of any analgesia resulting after a lapse of ten to fifteen minutes which was the recovery time from the ether alone. Some of these animals were observed over a period of sixty days, and were sacrificed at intervals to obtain the nervous systems for pathologic examination. Forty-five autopsies were performed on the animals receiving procaine, vitamin B_1 and thiamin-procaine. No signs of gross pathologic conditions were noted in the brains or spinal cords or their coverings, except distention of the blood vessels overlying the brain in those animals dying soon after convulsions produced by excessive doses of vitamin B_1.

To the first group of fifty-one cats, fifty-nine injections of procaine hydrochloride alone were given in doses of 7.5 to 45 mg., and in concentrations of 0.75 to 11.66 per cent. Usually an amount of cerebrospinal fluid was withdrawn equal to the amount of solution of the drug to be injected. Most of these cats survived injections up to 20 mg. With 20 mg. or more, respiratory paralysis resulted in death in two and a half to twenty minutes; in each case the heart continued to beat for some time after respiration had stopped. A lethal dose of 77 per cent was found for 20 mg. of procaine hydrochloride (thirteen cats), while a lethal dose of 83 per cent was found for 30 mg. (eighteen cats). It was evident that 15 mg. was about the maximum safe dose for cats intracisternally.

Anesthesia from the ether having worn off, the animals were tested for pain sensation and reflex muscle contractions by pinprick. Analgesia lasting over fifteen minutes was not observed, except in those occasional cats surviving after administration of 25 mg. or more. Undoubtedly it is the final concentration of the procaine in contact with the respiratory centers which determines whether the animal will live or not, and this depends upon the amount of cerebrospinal fluid removed, the amount left behind, the volume of the solution injected, and probably intrinsic factors such as the state of general health and vitamin nutrition of the animal.

In two monkeys included in this group, injection of 20 mg. of procaine hydrochloride cisternally resulted in death in four and six minutes, respectively.

A second group of fifteen cats were given sixteen injections of thiamin chloride alone intracisternally, in doses of 1 to 90 mg. Definite stimulation of respiration was noted with doses over 10 mg., and marked stimulation with doses over 20 mg. which resulted in periods of hyperventilation, alternating with periods of apnea; general convulsions finally led to death from exhaustion. These convulsions followed doses of 15 to 90 mg., some animals surviving repeated generalized convulsions for over one hour; others died after convulsions lasting from forty minutes to over three hours. With the largest dose of 90 mg. death came in five minutes before any convulsion could develop.

One monkey survived an injection of 40 mg. of vitamin B_1 cisternally, and a convulsion developed only after an additional injection of 80 mg. was given.

In the third group seventy-two cats received thiamin chloride combined with procaine hydrochloride in varying proportions. A small dose of 5 mg. of procaine was found to protect against a lethal dose of 90 mg. of thiamin, while 5 mg. of thiamin protected against a lethal dose of 30 and 35 mg. of procaine. In these combinations the procaine was increased to as much as 100 mg., while other injections contained as much as 110 mg. of the vitamin.

These experiments show that the toxicity of procaine is diminished by the thiamin chloride. In one case, five times the lethal dose (77 per cent) of procaine could be safely given combined with this vitamin. It is conservative to say that the maximum safe dose of procaine hydrochloride can safely be doubled if combined with vitamin B_1.

When more than 5 mg. of procaine were given with vitamin B_1, no convulsions developed, although some animals showed definite signs of respiratory stimulation. A combination in which the amount of vitamin B_1 was half the dose of procaine hydrochloride appeared to

give ample protection without undue respiratory stimulation.

In one monkey, 5 mg. of thiamin failed to protect against 20 mg. of procaine. Another monkey survived this combination, and subsequent administration of even twice the amounts. However, several days later this same animal succumbed in six minutes after 20 mg. of procaine alone were given.

A comparative study of the toxicity of some other spinal anesthetic drugs was made in a similar manner. The drugs tested and their relative toxicities causing respiratory paralysis after cisternal injection in cats were as follows:

Procaine hydrochloride—arbitrary standard =	1
Pontocaine® (Winthrop)*	40
Nupercaine® (Ciba)	40
Cocaine hydrochloride (Merck)	4
Stovaine (Poulene Frères)	4
Metycaine® (Lilly)*	3
Tropococaine hydrochloride (Merck)	2
Tutocain® (Winthrop)	1.5
Apothesine® (Parke, Davis)	1.5
Novocain® (Winthrop)	1
Neocaine (Anglo-French)	1
Aminocaine® (Seydel)*	0.5

* The toxicities of pontocaine, metycaine and aminocaine were *increased* by vitamin B_1.

Procaine hydrochloride combined with thiamin chloride proved to be the safest combination, allowing for the largest amount of anesthetic drug to be given. The effectiveness of such a combination was then tested clinically for spinal anesthesia for various major intra-abdominal and pelvic operations. As much as 250 mg. of procaine hydrochloride with 125 mg. of thiamin chloride given by lumbar injection produced satisfactory anesthesia, and in many cases no ephedrine or supportive measures were necessary. A proportion of two parts procaine hydrochloride and one part thiamin chloride in solution put in sterile ampules without any stabilizer or preservative was found to give satisfactory anesthesia four years after its preparation.

When thiamin was added to procaine for local anesthesia, the anesthetic properties were found to be augmented.[33–35] After experimental and clinical research, Bazzocchi and Bocchetti in Italy found that by adding 15 to 30 mg. of vitamin B_1 to 60 to 100 cc. of anesthetic solution, the quantity of anesthetic drug could be remarkably decreased to about one-third or one-half the usual amount. Thus it was possible to operate upon extensive hernias using 100 to 200 mg. of novocain solution to which vitamin B_1 had been added. They stated the obvious advantages of decrease of general toxic action, attenuation of local toxic effect, and the possibility of infiltration anesthesia of far greater areas than usually possible.

They also noted a reinforcing action of vitamin B_1 in spinal anesthesia. The introduction of a spinal anesthetic, to which this vitamin had been added, permitted reduction of the average dose of the solution to one-third, while the same anesthetic effect was obtained. Neither deleterious effects nor troublesome headaches, which occasionally follow spinal anesthetics, were observed. The mechanism through which vitamin B_1 exerts its reinforcing action on anesthetics was not explained, but they believed the vitamin improves and facilitates the absorption of the anesthetic by the nerve structures. Vitamin B_1 alone had no anesthetic action.

It is interesting to note that vitamin B_1 also lessens the toxicity of procaine intravenously. For example, a patient who could tolerate only 300 cc. of dilute procaine solution before he became stuporous was able to take 500 cc. on two subsequent occasions when vitamin B_1 was added in half the amount of procaine. Satisfactory general analgesia was obtained without any undesirable effects.

COMMENTS

The comparative toxicity of a number of spinal anesthetic drugs was determined by noting the smallest amounts which caused respiratory paralysis and death after direct injection into the cisterna magna of cats. Because thiamin chloride was found to stimulate respiration specifically when introduced alone via the same route, this vitamin was added to the various drugs tested to determine if it would overcome their toxic effects. Some anesthetics were made more toxic by this vitamin, others less toxic. Of the latter, procaine hydrochloride combined with thiamin chloride proved to be the safest, allowing for the largest amount of anesthetic drug to be given with safety. It was found that several times the lethal dose of procaine could be given safely to cats intracisternally if combined with vitamin B_1.

Clinically, the combination of procaine hydrochloride and thiamin chloride in propor-

tion of 2:1 has been given in doses as high as 250 mg. of procaine and 125 mg. of thiamin, with satisfactory anesthesia and no untoward effects. Several times the customary maximum dose of procaine may be found safe to use clinically if combined with this vitamin.

Others have reported that vitamin B_1 appears to augment the anesthetic action of procaine when used for spinal or local anesthesia in smaller dosages than usual. For more extensive procedures requiring more prolonged anesthesia, the use of much larger dosages of procaine than is customary, combined with vitamin B_1, should be found of practical value.

The combination of thiamin and procaine increases the scope of use for this anesthetic drug, particularly for intravenous therapy.

SUMMARY

1. Thiamin chloride (vitamin B_1) detoxifies procaine hydrochloride when given intraspinally, intravenously or by infiltration.

2. Thiamin chloride augments the anesthetic action of procaine hydrochloride.

3. These unique findings permit the use of smaller doses of procaine hydrochloride for ordinary procedures, and larger doses for more prolonged and extensive anesthesia.

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Solitary Bone Cysts

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Although most of us have had some experience with the solitary bone cyst, it is a less common disorder than one would believe.

Fig. 1. Case 11. Healing of a cyst over a four-year period following x-ray therapy.

In an active bone clinic at a large hospital only fifty-two cases were collected in a twenty-six-year period. Larger series have been reported but individual experience must necessarily be small.

In the past few years the literature on this subject has either been neglected or skillfully avoided, hence the decision to review the experiences of others and add a few cases of our own.

Terminology. Historically, the description of a solitary bone cyst in modern times is credited to Virchow in 1877. It is important to clarify this term. The old one, localized osteitis fibrosa cystica, is unsatisfactory and should be discarded. This name predates the discovery of hyperparathyroidism as the cause of multiple osteitis fibrosa of von Recklinghausen, and was thought to be a solitary cyst similar to the multicystic disease. The two conditions have dissimilar etiology and pathogenesis. In spite of this, McWhirter as recently as last year used the term to identify an intermediary developmental entity between giant cell tumor and solitary bone cyst.

The word unicameral has entered the literature representing the single-chambered nature of these cysts. This term is descriptive but provocative, and because of trabeculation of the walls of the cyst, it is open to argument unless proved at operation. The term dysfibroplasia is also controversial, as are others. Hence, we still prefer the simple term of solitary bone cyst.

Classification. No complete classification exists. However, the terms active and latent have been described by Jaffe and Lichtenstein.[5] Active refers to lesions near the epiphyseal plate which are potentially able to grow, and latent refers to those distant to the plate. In view of the reports of Stewart and Hamal, and Grahams, this classification has considerable significance in choice of treatment.

Etiology. As with all diseases of obscure etiology, there are a number of theories, each

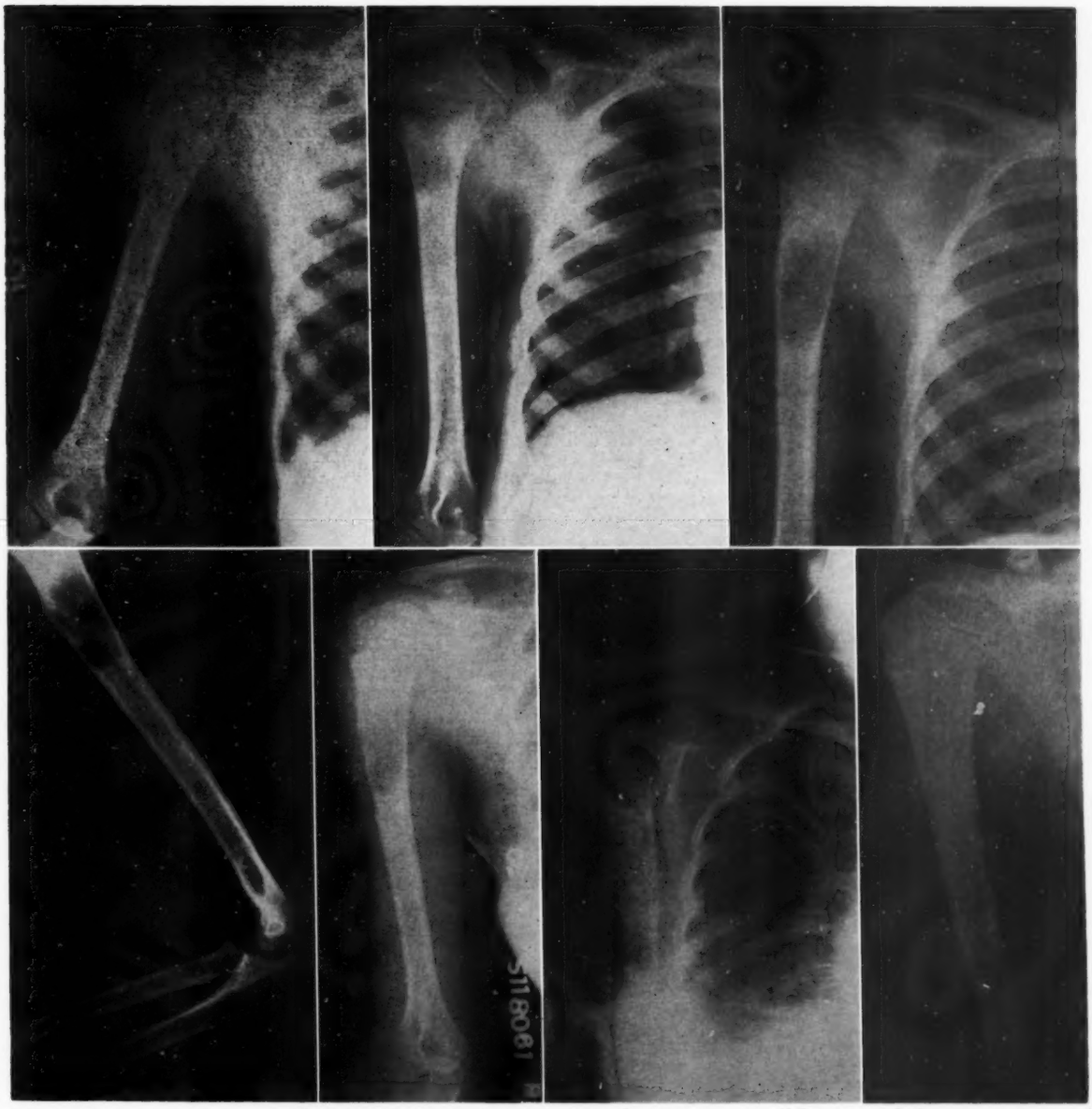

FIG. 2. Case IV. Active unstable cyst. Conservative treatment for one year until latent and then curettage and chips with successful obliteration of the cyst.

with its authoritative proponent. It is not our intention to become involved in an academic discussion so they merely will be mentioned and one will be elaborated upon.

1. Traumatic: A non-absorbed hematoma is the site of cyst formation (Pommer)
2. Inflammatory: Low grade infection causes the cyst
3. The cyst is an osseous dystrophy (Jaffe restating Mikulicz[5])
4. The cyst is the healed stage of a giant cell tumor (Geschickter and Copeland[3])
5. Metabolic: Faulty calcium metabolism
6. Bone tumors with cystic softening
7. Abnormal hyperplasia of osteoclasts due to trauma
8. A recent discussion which is by no means original tries to tie in giant cell tumors, osteitis fibrosa cystica and solitary bone cysts under one etiologic theory. It suggests that these three conditions are variants of the same process and the use of the term dysfibroplasia is offered.

FIG. 3. Case v. Illustrates uncommon place for cyst in a flat bone. The fracture is evident through mandible.

If a developmental error occurs late, just before closure of the epiphysis, the fibrous tissue remains in an area with a rich blood supply and does not undergo degeneration. The blood vessels become dilated due to the lack of fibrous support and blood flow through the lesion is increased so that the lesion may even pulsate and erode bone. As the lesion enlarges bone debris forms cells which stimulate a giant cell reaction. This results in a giant cell tumor. If the error occurs in an intermediary stage, the circulation is not good enough for giant cell tumor and localized osteitis fibrosa cystica results. If the error occurs early, the circulation is poor and degeneration of the area takes place, leaving a solitary bone cyst.

McWhirter's statement, however, that this explains why cysts occur most frequently in the mid- and lower humerus is extremely disturbing, for in the American literature and in our own experience the site of predilection is in the upper humerus.

Although we cannot subscribe to a theory which works in Scotland and not in this country, there certainly is merit in the thought that giant cell tumors and bone cysts are related in pathogenesis.

Diagnosis. The diagnosis is not difficult and more than 60 per cent of the cases become evident when fracture through the cyst occurs and a roentgenogram is taken. The others are found accidentally when roentgenograms are taken for other reasons. In a small percentage, symptoms such as discomfort and limp may occur and in very large cysts some deformity may be detected on physical examination, especially in the humerus.

Differential Diagnosis. Although malignancy, Brodie's abscess, gumma, enchondromas and von Recklinghausen's disease must be considered, these can be ruled out with simple examinations and the clinical course.

Differentiation from giant cell tumor may be more difficult and a few differences might be mentioned.

Solitary bone cysts are a disease of the first two decades; giant cell tumors usually occur in the second or third.

The solitary cyst almost invariably occurs on the metaphyseal side of the epiphyseal plate. One of the exceptions is Hutter's case in which part of the epiphysis in the upper tibia was also involved.[4] Giant cell tumors begin in the epiphysis.

Solitary cysts are usually symmetric whereas giant cell tumors enlarge asymmetrically.

In cysts the cortex is usually intact, whereas in giant cell tumors the cortex is often involved and perforated.

Finally, the pathologic examination of the tissue, if surgery is performed, is further evidence of the type of lesion with which we are dealing.

REVIEW OF LITERATURE

In 1942 Allredge[1] reviewed 152 cases of fibrocystic disease of bone and concluded that treatment of the fracture alone was unsatisfactory. He also wrote on the dangers of radiation treatment, and in 1941 reported two cases in which malignancy developed after radiation. At any rate, danger to the epiphysis and the question of malignancy make radiation poor treatment. Allredge concludes that the proper form of treatment is: (1) resection of nonessential bone; (2) curettage and bone chips, if in the epiphysis; (3) massive grafts if extensive and in the diaphysis.

James, Coley and Higinbotham reported twenty-six cases from the Memorial Hospital, eleven of which had pathologic fracture.

FIG. 4. Case VI. Fracture through an active unstable cyst with eventual obliteration with conservative treatment.

Eleven patients had simple curettage, fourteen had curettage and bone chips or other grafts and one had excision of a rib. Eighteen results were considered good. Two cysts healed incompletely. Six cases recurred, requiring two and three operations before healing occurred. The authors conclude that the treatment of choice is curettage and a bone graft. They also refer to two cases treated by irradiation with malignancy thirteen and fifteen years later, respectively.

Stewart and Hamal of the Campbell Clinic reported on forty-six cases treated in three groups. In the first group (fourteen cases) no treatment was given except for pathologic fracture when present. Eight patients had fracture and of these, six had excellent clinical results with partial obliteration of the cyst. One result was poor and one was not followed long enough. Of the cases with no fracture, one healed spontaneously, three remained unchanged and two were not followed. In the second group irradiation alone was used on three cases with two excellent results and one poor one. In the third group curettage, cauterization and bone chips were used in various combinations.

In the latent phase all but one patient showed excellent results. Of ten in the active phase, only *one* showed excellent results after one procedure. They concluded the following: (1) resect non-essential bone and (2) do not operate on those patients with lesions in the active phase. The authors believe that reluctance to curette adequately near the epiphysis of a child leads to an unsuccessful procedure. Their

FIG. 5. Case VII. Fracture of the posterior lip of the tibia with no disturbance of the stable cyst. Some authors prefer the term of localized osteitis fibrosa cystica for this type of cyst.

conclusion is that the procedure of choice is curettage, cauterization and chips.

Graham reviewed thirty-one cases at the Hospital for Joint Diseases. There was an overall recurrence rate of 41.9 per cent; after curettage and bone chips alone the recurrence rate was 60 per cent. Recurrences were more common when operation was performed in the active stage.

Two cases of epiphyseal growth disturbances were noted in patients operated upon two

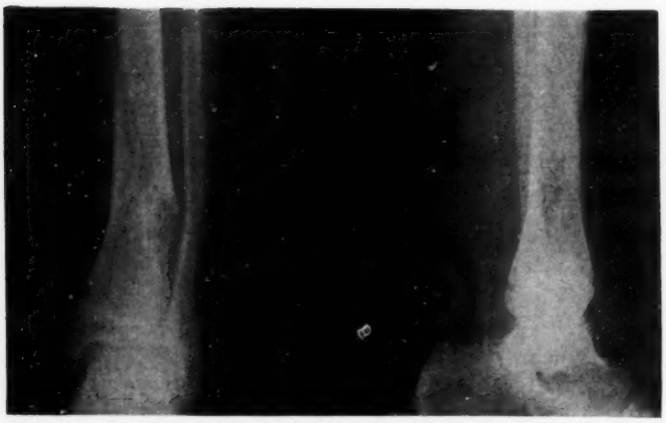

FIG. 6A. Case VIII. Fracture through a stable cyst with healing. Surgery unnecessary.

FIG. 6B. Case VIII. Healing of fracture. Cyst still evident.

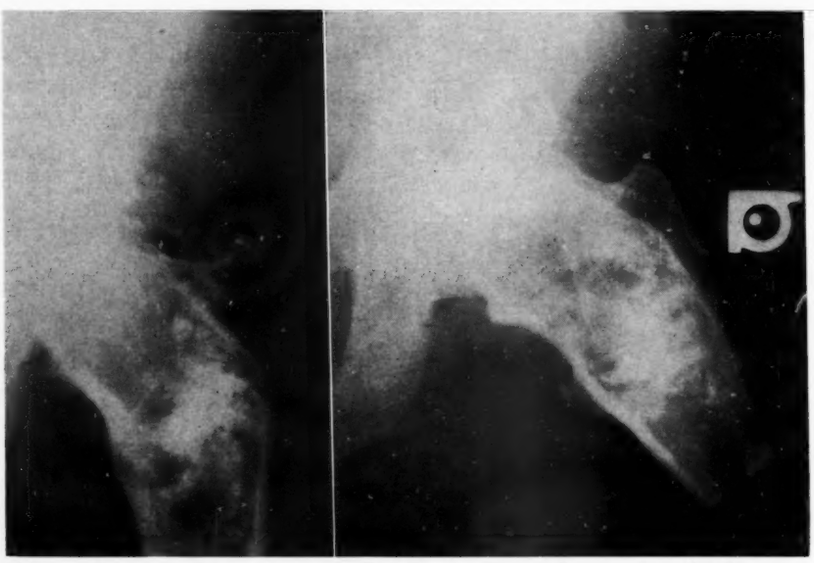

FIG. 7. Case IX. Cyst in the so-called "active" stageknown to exist for more than twenty-five years.

and three times. In one case peroneal nerve palsy followed operation for cyst of the fibula head.

OBSERVATIONS

It is obvious from the previous reports that surgery for the obliteration and cure of these cysts leaves much to be desired. (Even irradiation has given better results, but the dangers mentioned far outweigh its value.) The recurrence or poor result rate exceeds 33 per cent in the best series reported by James, Coley and Higinbotham and many of their cases have not been followed long enough. Cases have been reported as having recurred as long as eight years after supposed surgical cure. After examining the treated and untreated results of Stewart and Hammal, and taking into account the stage of activity, we wonder whether a better set of indications could not be worked out, on the basis of stability.

In our Case VII (Fig. 5), for example, a boy was subjected to trauma severe enough to fracture his posterior malleolus, yet a nearby bone cyst remained intact. It is believed a good result could be anticipated if we operated on the cyst, but is it necessary in view of stability?

In Case VIII (Fig. 6) fracture occurred through the lower tibial cyst. The end result of treatment of the fracture alone has resulted in what we would term a stable cyst area.

In Case IX (Fig. 7) we had evidence of a cyst

existing in the "active" stage for more than twenty-five years without mishap. It is true that this man is fortunate not to have sustained a fracture and not to have symptoms but we wonder what results elective surgery would have given us in this case.

These observations have led us to suggest the following classifications for treatment. This is intended to be flexible, and where stability ends and instability begins must be left to the good judgment of the individual surgeon. Certainly the same cyst will be less stable in a weight-bearing limb than in the arm. If the diagnosis is in question, certainly operation should be performed if only to provide a biopsy.

SOLITARY BONE CYSTS

I. Stable
   A. Latent (Cases VII and VIII)
      1. Advise no treatment except for fracture if it occurs
   B. Active (Case XI)
      1. Advise no treatment except for fracture if it occurs
II. Unstable
   A. Latent (Case X)
      1. Advise radical surgical procedure electively
         a. Curettage, chips, grafts
         b. Resection and double-onlay graft with chips in severe cases
   B. Active (Cases IV and VI)
      1. Advise conservative procedure
         a. Non-surgical
         b. Surgical

In this last group the aim should be to increase stability rather than to be concerned about complete obliteration at the risk of damaging the epiphysis. Key (at Obers' suggestion), in an attempt to perform a more complete operation, curetted through the cyst to the marrow cavity and still had recurrences. It is this group which is most problematic, and the basis for treatment should be supported until the cyst migrates to a latent position and has a greater possibility for cure. If the cyst does not migrate, a multiple-stage procedure should be anticipated (especially if the growth is in the lower limb) until the area gains sufficient stability.

The classifications of latency and activity refer as much to position as to ability to grow. Many cysts, as in our Case IX, have been in the so-called active phase for more than twenty-five years, and one hardly thinks of activity as being present. Nonetheless, on the basis of anticipated results, it is useful to maintain this classification as an index of proximity of the cyst near the epiphyseal plate as much as the potentiality of growth.

FIG. 8. Case X. Fracture through an unstable cyst. Ideal for curettage and grafts.

CASE REPORTS

CASE I. W. P., a seven year old boy sustained a pathologic fracture through a humeral bone cyst. Conservative treatment resulted in healing in four weeks. The cyst has persisted, but neither disability nor refracture has occurred in a four-year follow-up.

CASE II. D. G., aged sixteen, sustained a pathologic fracture through a humeral bone cyst on October 13, 1943. On December 27th there was evidence of enlargement of the cyst. In January, February and March, 1946, this patient received x-ray therapy. The last x-ray on July 17, 1947, showed incomplete but progressive obliteration of the cyst. (Fig. 1.)

CASE III. Patient B. G., sustained a pathologic fracture through a humeral bone cyst on March 2, 1952. X-ray therapy was given. One year later the cyst was partially obliterated and the fracture well healed.

FIG. 9. Case XI. Intertrochanteric fracture through cyst with healing after open reduction and curettage.

CASE IV. M. G., a five year old boy, sustained a pathologic fracture through a humeral bone cyst on October 19, 1950. He was treated conservatively, and x-ray one year later showed excellent healing but persistence of the cyst. Figure 2 demonstrates the cyst immediately before operation. Saucerization, curettage and insertion of bone chips was performed on November 6, 1951, and the last illustration demonstrates the end result—complete obliteration of the cyst. This patient was treated conservatively in the active phase and radically when the cyst became latent.

CASE V. M. K., a forty-three year old man, had a solitary bone cyst in the mandible. This was a rather unusual site, for bone cysts occur least frequently in the flat bones. The patient was treated with intraoral splints and a Barton bandage. Surgical eradication of the cyst was refused. (Fig. 3.)

CASE VI. Patient M. P., aged nine, sustained a pathologic fracture through a large cyst in the upper femur on December 24, 1948. Four months later healing was complete. Six months after fracture a tendency toward obliteration of the cyst was noted, as well as migration away from the epiphyseal plate. Ten months after fracture obliteration became more complete, and five years later (Fig. 4) almost complete obliteration of the cyst was noted, as was migration away from the epiphyseal plate.

CASE VII. L. A., a nineteen year old boy, was injured while playing football and sustained a fracture to the posterior malleolus of the tibia. Above this was an intact solitary bone cyst. The fracture healed well. This case is significant in that the nearby cyst was undisturbed; hence, it was stable. (Fig. 5.)

CASE VIII. J. M., a thirteen year old boy, was injured while playing football on October 10, 1948. He sustained a fracture through a solitary bone cyst in the lower tibia. At three months there was union, and ten months later there was complete union but persistence of the cyst. (Fig. 6.)

CASE IX. L. S., a fifty-three year old man, had a cyst which had been present for more than twenty-five years. This cyst was discovered on routine x-rays. When his original physician was contacted for further history he was able to describe x-rays taken twenty-five years previously identifying the existence of the same cyst. (Fig. 7.)

CASE X. R. J., a seventy-four year old man, was admitted with a pathologic fracture through the upper tibia. The skin on the upper aspect of both legs was completely denuded. This resulted from severe burns received while stamping out a fire the year before. In an attempt to clear up his skin so that the cyst and fracture could be attacked surgically, the infection in the skin was brought under control. Skin grafting was started, after which the patient promptly removed his dressings and scratched off the skin grafts. It was elected to treat him conservatively and Figure 8 shows moderately good healing after seven months, at which time he was ambulatory and returned home. We have not heard from him since. This case would have been ideal for curettage and grafts, being latent and unstable.

CASE XI. H. C., a nine year old boy, sustained an intertrochanteric fracture through a bone cyst after a fall on his hip. Open reduction was performed a few days later, the cyst area was curetted and three wires were used for fixation. X-rays showed the beginning of union at three months, and after nine months x-rays showed complete union with almost complete obliteration. (Fig. 9.)

All of these patients had bone survey and chemical studies and in no instance did we find other cysts or disturbances in calcium metabolism.

It is our belief that stable cysts should not have elective operation. Only treatment of the fracture, when it exists, is necessary. Furthermore, unstable cysts in the shaft should have vigorous treatment, and those near the epiphysis should receive conservative treatment until they become latent. Conservative treatment may be non-surgical or less radical surgery, even if in multiple stages.

SUMMARY

1. Solitary bone cysts are reviewed and eleven new cases are reported.

2. A classification for treatment based on stability is suggested.

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Surgical Management of Diverticulitis

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THE most significant trend in the modern management of diverticular disease of the colon is carefully planned surgery undertaken prior to the development of frequently disastrous complications. A sigmoid colon studded with diverticula undergoing recurring episodes of inflammation should be resected. This attitude is thoroughly sound. Resection of such bowel is no longer the hazardous procedure of a few decades ago, and the old custom of watchful waiting is rapidly becoming obsolete.[1,4,5,10,13,21,24]

The principle of preventive surgical intervention although valuable and important (and in our experience a safe approach), still falls short of solving many problems manifested by diverticulitis. The single flaw in its application is the fact that the diagnosis is not made in many cases until a serious complication ensues. Furthermore, even when the diagnosis has been established, evaluating symptoms, interpreting radiographic studies and deciding whether or not surgery is warranted or when it should be timed is indeed difficult. Few cases are clear-cut and they present indisputable indications for preventive operation. Our present criteria include repeated episodes of acute inflammation, evidence of chronic changes with narrowing of bowel lumen, progression of the disease to involve a greater extent of bowel and evidence of hemorrhaging. Often a single severe attack is sufficient indication for preventive resection after symptoms have subsided.

Since surgical complications not uncommonly precede even the suspicion of diverticulosis or diverticulitis, our approach to the subject will be to consider these mishaps in detail. Most important toward successful management is the realization that all surgical measures short of resection of diseased bowel are futile. Resection should be the ultimate goal, and emergency intervention or palliative procedures merely prepare the patient for resection.[13]

PERFORATION

A not uncommon question asked at medical meetings and round-table discussions is: How should acute perforation of a sigmoidal diverticulum be managed? Before an intelligent answer can be given information must be obtained relative to the findings prior to and at the time of exploratory laparotomy. Some surgeons have cautioned against any procedure other than colostomy, while others have advised primary repair of the perforation.[2,3,7,11,12,21] The difference in viewpoint arises mainly from experiences with several essentially different sets of clinical findings.

The sudden rupture of a diverticulum, which is by no means rare, may occur after a short but acute attack of inflammation. It may also occur without inflammation as a complication of diverticulosis.[9] Contents of the bowel leak from the perforated area into the general peritoneal cavity. Surrounding intestine and omentum have not had time to wall off the process and peritonitis becomes generalized. In these patients the perforated bowel may be easily mobilized; and because the wall of the bowel is not particularly edematous or friable, primary closure is suitable.

In other instances peridiverticulitis with adhesions to other viscera, epiploic fat tabs and omentum surrounds the acutely inflamed intestine. When perforation does take place, partial walling-off of the process has been in progress and upon exploration of the abdomen an inflammatory mass may be found from which purulent material and fecal matter exude. Here primary closure is quite another consideration and usually is out of the question. Even if the involved bowel can be liberated its wall is friable, edematous, congested and covered by purulent exudate. Suturing usually produces a second opening larger than the first.[20] The danger of spreading infection is great so that rather than unsuccessfully attempting to dissect into the mass, the area should be disturbed as little as possible.

Perforation may not always penetrate into the abdominal cavity. Often it is retroperitoneal or ruptures into some other viscus. If

retroperitoneal, an abscess forms followed later by a fistula.

TREATMENT OF PERFORATION

Primary Closure. The diagnosis of acute perforation is rarely made until the abdomen is explored. Usually acute appendicitis, perforated peptic ulcer or some other surgical emergency is suspected. Feces and purulent material are discovered and if peridiverticulitis is minimal, there may be little or no inflammatory mass. Search for the perforation must be attempted. It may be hidden by epiploic fat or loosely adherent to small intestine. Once the ruptured diverticulum is discovered and the surrounding bowel wall is found to be fairly normal it can be quickly repaired. We prefer interrupted sutures of No. 000 chromic catgut or No. 35 atraumatic® alloy steel wire. If possible, the defect should be closed transversely. The abdominal cavity is thoroughly irrigated with saline solution until all gross contamination has been removed. Transverse colostomy may or may not be performed. Its indication depends upon the presence of inflammatory involvement at the suture site.

Exteriorization. Often moderate to severe inflammatory changes involve the bowel surrounding the site of perforation. Its wall is indurated, friable, edematous and thickened so that suturing is out of the question. In such circumstances exteriorization is the treatment of choice. Several methods may be employed. The colon is mobilized, and either the loop containing the perforation is brought out through the abdominal wall or the bowel is divided at the site of perforation and both segments are exteriorized. The latter procedure provides complete defunctioning as the proximal stoma functions as a colostomy. The distal stoma can be irrigated. In the former procedure the loop of bowel is prevented from retracting by partly dividing its mesocolon so that fascia may be approximated. A glass rod or a rubber tube may be passed under the loop to hold it in place. A large-sized rectal tube or mushroom catheter is passed through the perforation into the lumen and sutured in place. The abdominal cavity is thoroughly irrigated with saline solution before closing.

Transverse Colostomy and Simple Drainage. If a large inflammatory mass is found at the site of perforation, which prevents demonstration of the rupture, and mobilization of the involved bowel is impossible, transverse colostomy is performed. The abdominal cavity is thoroughly irrigated to remove gross contamination and a Babcock sump drain is placed adjacent to the infected tissues.

Antibiotics. In each of the previously mentioned methods antibiotics play a role of signal importance. One of the most useful of the newer technics is the injection of a neomycin solution directly into the colon well proximal to the site of perforation. A No. 16 or 18 needle is inserted into the bowel and attached to a 50 cc. syringe barrel (the piston has been removed). A solution of 5 gm. of neomycin in 500 cc. of sterile saline is now poured into the syringe barrel and thus into the bowel. The site of injection is repaired by a small purse-string suture of fine silk. The solution is flushed through the entire length of the colon by manipulation, care being taken to avoid excessive pressure at the site of perforation. This procedure results in a rapid reduction of the intraluminal bacterial flora so that further leakage from an unrepaired or unexteriorized ruptured diverticulum is rendered less hazardous. It is an excellent safety measure even though repair of the perforation has been successful or after exteriorization has been accomplished. A solution of 1 gm. of streptomycin and 500,000 units of penicillin is instilled into the abdominal cavity through a Babcock sump drain after closing the abdomen. Penicillin and streptomycin are then continued parentally in the postoperative management.

Definitive Treatment. Resection of the involved bowel must be considered after infection and inflammatory involvement have subsided. Usually a period of six weeks or more is required contingent upon the circumstances.

FISTULA

Fistula is a sequela of perforation. It is frequently associated with abscess formation and considerable inflammatory reaction. The pathogenesis has its onset in the slow perforation of a diverticulum in the midst of peridiverticulitis with adhesions to an adherent viscus or abdominal wall. In a majority of cases abscesses form and penetrate through surrounding tissues to perforate bladder, kidney, other intestine, vagina, rectum, Fallopian tubes or abdominal wall. Purulent material may penetrate into retroperitoneal areas, particularly those in more dependent locations such as retrorectal,

presacral and supralevator spaces. Upon exploring the abdomen in the case of diverticular fistula, considerable chronic inflammation is encountered and single or multiple abscesses may be ruptured in the process of mobilizing the infected bowel and fistulous tract.

TREATMENT OF FISTULA

No plan of surgical management is satisfactory unless it includes a complete resection of the diverticula-studded diseased bowel. The most conservative and frequently the safest approach[17] is a procedure consisting of three stages: (1) transverse colostomy, (2) resection of diseased bowel with removal of fistula and repair of involved viscus, (three weeks to three months after first stage), and (3) closure of colostomy. The disadvantages of this method are the several operations required and the inconvenience of colostomy. The second alternative is primary resection of the bowel with repair of fistula in one stage.

In making a decision between these two methods, it is necessary to consider the safety factor combined with the three-stage operation.[24] A well patient is the goal to be achieved. The primary resection procedure has become more popular as a result of the antibiotic control of fecal bacterial flora. Our experience, however, has led us to select the stage operations in the face of active inflammatory processes. If infection is minimal, primary resection is then the choice. In evaluating the individual patient, history, physical findings, sigmoidoscopic and roentgenographic studies are most important. In addition, the patient's temperature is observed carefully over a period of five days to a week. If it is elevated, colostomy is the best approach. Additional significant findings are white blood count, differential count and sedimentation rate.

We seldom perform complimentary transverse colostomy at the time of primary resection as we believe that if such is indicated, then primary resection is ill chosen. Nothing is lost by performing preliminary colostomy and later resecting the infected tissues after inflammation has subsided.

STRICTURE

The special problem presented by stricture accompanying diverticulitis is the ruling out of carcinoma. The frequency with which this is associated with diverticulitis is well known.[18–20,24] Much can be learned from radiographic appearances, but there is no certainty until the suspicious segment has been resected.

Stricture due to diverticulitis is tubular in type and denotes persistent exacerbations of acute, subacute and chronic inflammation. Rarely is the segment of narrowed bowel found free of intramural abscess formation, ulceration, edema and areas of necrosis. Total obstruction is not very common, and when present is usually associated with peridiverticulitis and adhesions to small intestine with the presence of a large inflammatory mass. Partial obstruction may become complete when inflammation is acute and associated with edema and mural induration. This is rather dramatically relieved by diversion of the fecal stream and defunctionalization. Associated small bowel obstruction must be suspected in every such problem.

TREATMENT OF STRICTURE

As in dealing with the problem of fistula, a choice must be made between the three-stage procedure of colostomy, later resection and closure of colostomy, and primary resection without colostomy. Preoperative fever, elevated white cell count, rapid sedimentation rate and other systemic manifestations of infection indicate preliminary colostomy. Primary resection may be considered for cases in which there is incomplete obstruction and infection is minimal. The great advantage of primary resection is the earlier removal of possible carcinoma.

Obstruction often adds considerable difficulty in management. Of first importance is a correct diagnosis. Too frequently the small intestine is involved in peridiverticular infection and the patient's obstructive symptoms are due to blockage of this bowel and not to the tubular stricture. In such a situation transverse colostomy offers nothing. Exploratory laparotomy will reveal the true condition as the colon is not nearly as distended as the small bowel. Preoperative scout films of the abdomen are requisite, and a thin suspension, low pressure instillation of barium through the rectum aids in diagnosis.

Obstruction of the involved colon at the site of stricture will often vanish after defunctioning the colon by colostomy; in some instances we have observed the colostomy actually being closed later. This is to be con-

TABLE 1
MANAGEMENT OF DIVERTICULITIS (SEPTEMBER, 1940 TO NOVEMBER, 1955)*

Type of Colic Resection	Number	Deaths (%)
Resection with primary open anastomosis	44	2.2
Partial cystectomy with colic resection, open anastomosis	7	0
Partial cystectomy, sigmoidectomy, with preliminary colostomy (three-stage)	4	0
Ileocolectomy, abdominoperineal proctosigmoidectomy for associated adenocarcinoma of cecum	1	0
Mikulicz-type resection (1940 to 1948)	7	0
Abdominoperineal proctosigmoidectomy with preservation of sphincters	6	0
Left hemicolectomy with transplant of transverse colon to perineum, preservation of sphincters	1	0
Abdominoperineal excision (Miles) (associated carcinoma)	1	0
Sigmoidectomy with colostomy (Hartmann)	2	0

* Partial cystectomies: 11, or 15 per cent.

demned since the inflammatory process soon returns. Radical treatment is needed and should consist of complete resection of involved bowel after obstructive symptoms and evidence of active infection have subsided.

HEMORRHAGE

Once considered a rarity, hemorrhage is now known to complicate both diverticulosis and diverticulitis in enough instances to give it equal rank in incidence to obstruction, perforation or fistula. The bleeding is profuse and usually produces shock. Exsanguinations which resulted in deaths have been reported.[14,16,18] The blood may be bright red or dark in color with presence of clots. It occurs in diverticulosis as well as diverticulitis, and in our experience its incidence in the former has been greater than in the latter. The following criteria, according to Quinn and Ochsner,[18] should be fulfilled in order to justify the assumption that the bleeding is from diverticula: (1) the passage by rectum of gross blood, either bright or maroon in color; (2) barium enema evidence of diverticulosis or diverticulitis; (3) absence of other intrinsic lesions on rectal or proctoscopic examination; and (4) roentgenographic studies demonstrating the stomach and small bowel to be normal.

TREATMENT OF HEMORRHAGE

Most cases of acute hemorrhage will respond to conservative measures. Hospitalization with complete bedrest is requisite. Blood transfusions are quickly instituted to control shock. Sedation, low residue diet and parenteral doses of high potency vitamin K are administered. Should bleeding continue, surgical intervention is necessary and should consist of resection of the involved bowel even though in rare problems it means total colectomy.

In conservative management after hemorrhage has subsided, the patient is prepared for definitive resection of involved colic segments. To omit this is to invite further episodes of bleeding with perhaps disastrous results.

TREATMENT OF ACUTE, SUBACUTE AND CHRONIC DIVERTICULITIS

In considering the subject in its entirety we believe that in the absence of the surgical complications discussed previously, patients with acute, subacute and chronic diverticulitis should benefit from our accumulating evidence that preventive resection is justified.[1–3,21,23] The indications for such intervention must be thoroughly evaluated in each individual. Symptoms, history, physical examination, sigmoidoscopic study and barium enema examinations are fully appraised. Periodic studies are made every three to six months in those patients in whom preventive surgery is deemed unessential.

OPERATIVE RESULTS

Our experience with the management of diverticulitis from 1940 to November, 1955, encompasses a series of 189 cases. Of these, seventy-three underwent definitive treatment in the form of resection of diseased bowel with a mortality rate of 2.7 per cent. Fourteen patients of the 189 were treated by palliative operations with a mortality rate of 15.1 per cent. Conservative measures were begun in one hundred

one patients. Most of this treatment reflects the conservative approach advocated before 1950 of avoiding surgery except in patients with complications or suspicion of cancer. We do not have a complete follow-up on the patients not operated upon, but many of them were poor surgical risks and in an elderly age group in which surgery was deferred unless complications seemed eminent.

Table 1 indicates the methods of definitive resection undertaken and the mortality rate.

TABLE II
REHABILITATION (SEPTEMBER, 1940 TO NOVEMBER, 1955)

Number of patients resected	73
Known patients free of symptoms	51
Known number of patients with periodic distress (mild)	3
Known number of patients with periodic distress (severe)	1
Recurrent vesico-sigmoido-fistula	1
Number lost to follow-up	14
Completely rehabilitated (able to work)	54
Rehabilitation rate	94.7 per cent

REHABILITATION

The evaluation of any trend in surgical management cannot be fully appreciated without attention to returning the patient to good health and to those activities in which he participated before he became ill. In our series of resections follow-up studies were carefully made to determine the number of patients returned to health and occupations. (Table II.)

CONCLUSIONS

Re-evaluation of the entire subject of surgical management of diverticular disease of the colon indicates a greater need for preventive resection, particularly in the light of low mortality and morbidity now possible through the control of colic bacterial flora, improved measures in nutrition and electrolyte replacement.

Special problems will still persist despite preventive resection because in many patients the diagnosis of diverticulosis or diverticulitis is not made until a surgical complication develops.

Acute or sudden perforation of the colon can usually be managed by repair of the site of rupture. Transverse colostomy may or may not accompany this procedure. If the bowel wall is friable, local repair is not recommended. Exteriorization, if possible, is the treatment of choice. If the bowel cannot be readily mobilized, the inflammatory area is drained by a Babcock sump drain and transverse colostomy is performed. A neomycin solution (5 gm. in 500 cc. of sterile saline) is injected directly into the bowel lumen and flushed through the colon by gentle manipulation.

Fistulas are treated by resection of the diseased bowel and removal of the fistulous tract. The viscus penetrated by the fistula (in our experience usually bladder) is repaired. If a patient shows preoperative evidence of active infection and inflammation, a three-stage procedure is best planned consisting of transverse colostomy, resection and closure of colostomy. Three weeks to three months elapse between colostomy and resection.

Strictures are treated by resection. The ruling out of carcinoma is important, and because of its high incidence in diverticulitis primary resection rather than a three-stage operation is the procedure of choice if inflammatory reaction and infection are minimal.

Hemorrhage, usually severe and accompanied by shock, can be treated by conservative treatment in most cases. However, each such patient must be prepared for resection of diseased bowel when blood loss has been controlled.

Resection as a preventive measure in recurring episodes of diverticulitis can generally be undertaken in one stage. Prior to surgery the patient should be treated conservatively until acute infection, if present, has subsided.

The authors' series of seventy-three resections were accompanied by a low mortality rate of 2.2 per cent. In this series are eleven partial cystectomies undertaken for repair of vesical fistulas; no deaths occurred.

Palliative treatment has been outmoded and is rarely indicated unless other debilitating circumstances preclude definitive management.

Rehabilitation is excellent in a great majority of patients treated by resection provided a liberal segment of bowel is removed. Recently it has been our custom to perform left hemicolectomy with anastomosis of the distal transverse colon to the low sigmoid or rectosigmoid. Recurrence of symptoms is uncommon.

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Transvesical Prostato-adenomectomy with Primary Closure of Bladder

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HRYNTSCHAK[28–33] performed his modification of Harris'[19,20] transvesical adenomectomy in over 300 patients. Typically, on the first postoperative day the patient ambulates catheter-free, is not threatened by postoperative hemorrhage, empties the bladder completely and is dry at incision. This is a report of Hryntschak's procedure, performed in sixty-five unselected cases of prostatic hyperplasia.

METHOD

All patients had severe symptoms of prostatic enlargement ranging from weak stream with intractable pollakiuria to complete retention with or without overflow. All had a concentration of urea in the blood within range of intravenous pyelography; the latter was routine. Screening for latent carcinoma was by multiple perineal punch biopsy under local 0.5 per cent procaine hydrochloride. In patients with nodules open biopsy was done of the nodule and adjacent prostatic tissue by Belt's[3] subanosphincteric approach. We have not as yet encountered cancer with enucleated adenoma.

Debilitated patients were given blood preoperatively, even when hemoglobin concentration was normal, because of presumably contracted blood volume due to old age.[2,6,62]

The bladder was approached through Pfannenstiel's[49] incision, extending between the lateral borders of the rectus muscles. The rectus sheath was divided without being stripped of overlying fat. Pyramidalis and rectus muscles were separated in the midline after mobilizing the rectus sheath at the linea alba from underlying muscle up to the pubis and for 2 cm. cephalad.

The patient was placed in a tilted, head-down position so that the navel was lower than the pubis. The prevesical fascia was stripped toward the pubis and not away from it. Entry into the space of Retzius was thus avoided. The anterior surface of the bladder was generously mobilized from the peritoneum.

The bladder was entered according to Hryntschak.[32,33] The Walther bladder rectractor was used because it allows ample view of the vesical cavity without unnecessarily wide cystotomy. It is fenestrated and is composed of detachable blades.* The central blade was never used. After identifying the ureteral orifices and surrounding landmarks, the retractor was removed and the prostate enucleated.

Enucleation was performed without aid of rectal palpation in the first thirty-five cases. In later cases rectal palpation was employed, which we prefer because it: (1) pushes the prostate forward, (2) makes enucleation easier, (3) permits ready detection of residual adenoma in prostatic fossa, (4) maintains the correct plane and (5) allows bimanual pressure hemostasis immediately after enucleation.

The bladder retractor was reintroduced and gauze sponges were immediately packed into the prostatic fossa. Ureteral orifices were reidentified and a generous V-shaped wedge was excised from the posterior lip of the bladder (Fig. 1, *v*), so that the base of the bladder became almost flush with the posterior surface of the prostatic fossa.

The gauze sponges in the prostatic fossa were removed, and stay- and Z-sutures before ligation, Fig. 1, *z*; after ligation, Fig. 2, *x*) of No. 1–0 plain catgut were applied to the margins of the prostatic fossa at about 5 and 7 o'clock, by taking deep bites of 1 to 1.5 cm. These sutures were applied with long needle holders and No. 3 or 4 Mayo catgut needles,† which are strong enough not to break. If the posterior lip of the bladder was bleeding profusely, a Z-suture (Fig. 2, *v*) was applied. Any remaining spurters at the prostatic fossa

* V. Mueller & Co., Chicago, Ill., GU 1922.

† SN-10 of V. Mueller & Co., Chicago, Ill.

were clamped and suture-ligated. If the patient's arterial pressure was 20 mm. or more below his normal, it was returned to normal by transfusion and/or by sympathomimetic drugs, depending upon cause(s) of drop, before hemostatic suturing was considered complete. This principle was overlooked in one patient (VAH 33489), thus necessitating reopening of the bladder for more hemostatic suturing on the evening of the operative day.

A 24 F. rubber catheter (not latex) was introduced per urethra well into the bladder (Fig. 2, *c*) and fixed to the glans penis by a loose suture ligature of silk. This suture was applied with the penis maximally extended to avoid ensconcement of the catheter tip in the prostatic fossa. Transverse sutures of No. 1 plain catgut, one to three in number but usually one, were applied deeply across the prostatic fossa, as described by Hryntschak, to "pack off" venous oozing (Fig. 2, *t*.) The intravesical end of the catheter was then cut off elliptically 2 to 3 cm. from the margins of the prostatic fossa so that the surface of the ellipse faced anteriorly. (Fig. 3.)

The new Hryntschak catheter* has been used in the last fifteen cases. The catheter has an elbowed tip with one eyelet. The latter is at an optimal distance (6.5 cm.) from the symmetrically distensible balloon. The catheter is introduced after applying, but before ligating, the transverse suture of the prostatic fossa. The transverse suture is ligated over the tip of the catheter so as to include the catheter within the suture. The balloon is inflated in the prostatic fossa with 15 cc. of water. This keeps the catheter in place but is not great enough to prevent shrinkage of the prostatic capsule.

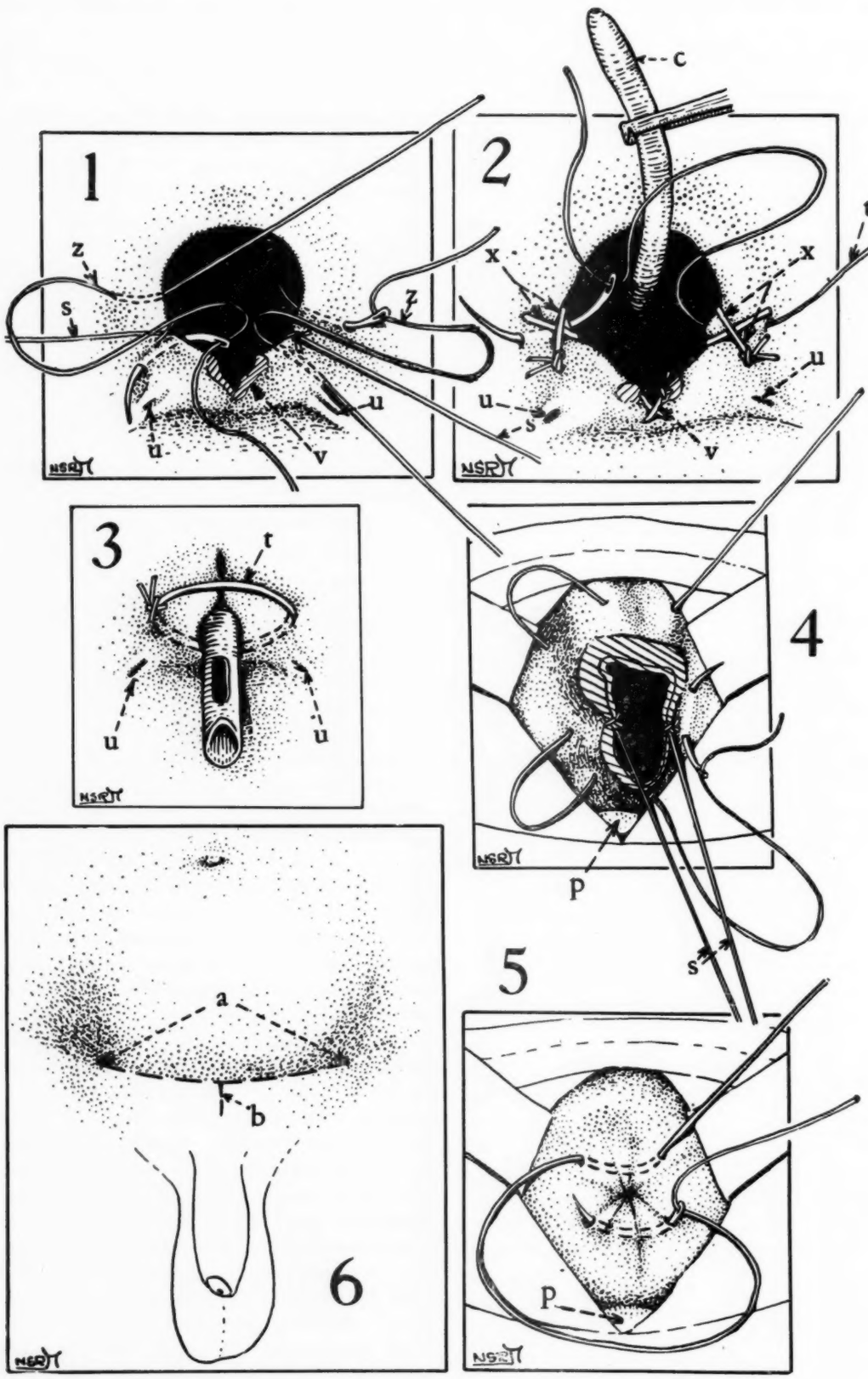
The bladder was closed by an inverting purse-string suburothelial suture of No. 1-0 chromic catgut. Correct application of this suture is essential (Fig. 4) and is aided by palpating the lining of the bladder with the index finger in the cystotomy. A No. 12 Ferguson needle† was used and four to five moderate bites around the cystotomy sufficed. (Fig. 4.) Hryntschak[32,33] has described application of this suture in detail. If our first attempt at producing an inverted purse-string suture was imperfect, we replaced it. This suture was usually reinforced with an overlying Z-suture. (Fig. 5.) Urinary leakage was thus prevented even when the bladder was allowed to become distended inadvertently to 400 cc. on the first postoperative day (VAH 38114). Valve action of the purse-string suture was demonstrated by readily introducing the tip of a closed forceps into the bladder lumen through the puckered dimple of the tightened suture although water would not leak out when injected into the bladder under moderate pressure. The bladder was irrigated with saline to test the adequacy of closure and hemostasis.

The rectus sheath was sutured with closely set interrupted No. 1-0 chromic catgut. A fine soft rubber drain was inserted to the bladder closure. Scarpa's fascia was apposed with running No. 3-0 plain catgut on each side of the drain. A 1 cm. cutaneous counterincision was made in the midline toward the pubis at a right angle to the main incision. (Fig. 6.) This small counterincision is important when the Pfannenstiel incision is used for prostatectomy with primary closure of the bladder because the transverse incision, being parallel to the lines of cutaneous tension, heals rapidly. Removal of the prevesical drain on the fourth or fifth postoperative day may leave a subcutaneous pocket of suppuration with prolonged drainage unless premature closure of the drainage tract is prevented by such a counterincision. We have not had such drainage in the forty-five or more cases in which this counterincision was used. The prevesical drain was sutured to the skin with silk.

If the catheter had been sutured to the glans penis, it was now also taped to the penis, thus preventing traction upon the suture. The patients never complained of pain from the suture when this had been done. The bladder was irrigated again in the operating room and the catheter left unclamped. The catheter was irrigated with about 30 cc. of saline every half hour until bleeding largely subsided; this usually occurred within six hours. The catheter was then irrigated every two hours. Slight to moderate suprapubic leakage occurred on the operative day in a few cases but was transient and inconsequential. Typically, the catheter was removed on the first postoperative day even when the urine was colored moderately with dark blood. The patients were able to empty their bladders readily and remained

* Made by Firma Rüsch, Würtemburg, Germany; and retailed by Metro Medical Distributors, Inc., Brooklyn, N. Y.

† SN-160 of V. Mueller & Co., Chicago, Ill.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dry suprapubically. Ambulation was on the first postoperative day.

Vasectomy was not routine and was usually restricted to poor-risk cases, in which it was performed before enucleation.

Preoperative medication was usually meperidine hydrochloride, 75 mg., and atropine sulfate, 0.4 mg., intramuscularly. Anesthesia was by spinal pontocaine or general ether-ethylene with ample oxygen.

RESULTS

Mortality. There were no operative deaths. One postoperative death occurred; this patient (VAH 46751) had moderate hypertension and his congestive myocardial failure was regulated by mercuhydrin® and digitoxin. His blood loss was slight and he had a smooth postoperative course. However, he died abruptly after a bowel movement on the second postoperative day, at which time he was voiding freely without catheter. Autopsy was not obtained. Immediately before death neuromuscular signs were consonant with massive pulmonary embolus or intracranial hemorrhage.

Postoperative hemorrhage occurred once (VAH 33489). The patient's bladder was closed while he was hypopiesic from anesthesia (systolic blood pressure 100 compared with his normal of 160 to 200) and when hemostasis thus appeared adequate. Upon return to the ward his systolic pressure was 200 mm. and he hemorrhaged. More hemostatic sutures were applied and his bladder was reclosed that evening. On the twelfth postoperative day he bled moderately but required no catheterization. At that time he was markedly hypoprothrombinemic.

Postoperative bleeding. A small amount of dark blood was usually lost during the first few postoperative days, not as clots but enough to give the urine a port wine color. This did not necessitate transfusion.

Postoperative fever. Most patients were given penicillin and streptomycin during the first five postoperative days. Maximal oral temperature varied between 98.6° and 102.2°F. (average maximal temperature, 100.6°F.) and usually occurred on the second postoperative day.

Two patients were febrile in the immediate preoperative period. The fever was attributed, after careful exclusion, to intraprostatic suppuration: (1) One patient (VAH 33634) ran a septic, severely febrile course after cystoscopy, with oral temperature up to 103.8°F. The gram-negative bacillus in his urine was resistant to antibiotics *in vitro*. A 90 gm. adenoma was enucleated on the third day after cystoscopy, under "cover" of intravenous drip of oxytetracycline (500 mg./L. 5 per cent dextrose in water). The maximal postoperative temperature was 102.2°F. on the day of surgery. The patient became definitively afebrile on the second postoperative day. On the fourth postoperative day epididymitis developed and the accompanying fever lasted two days. Three transverse sutures had been used across the prostatic fossa and the catheter was removed on the first postoperative day. (2) The second

FIG. 1. Prostatic fossa seen through the open bladder. (*s*) Stay-sutures through prostatic margin, right and left; (*u*) ureteral orifice; (*v*) wedge excised from posterior margin of prostatic fossa and apex of trigone; (*z*) Z-sutures, right and left, are being applied.

FIG. 2. Prostatic fossa seen through open bladder. (*c*) Tip of urethral catheter emerging through fossa and grasped in a toothed clamp; (*t*) transverse suture being applied; (*u*) ureteral orifice; (*v*) wedge excised from posterior margin of prostatic fossa and apex of trigone with a hemostatic suture (*x*); Z-sutures fully applied appear as X-sutures.

FIG. 3. Margins of prostatic fossa apposed about indwelling urethral catheter by application of transverse suture (*t*); (*u*) ureteral orifices.

FIG. 4. Application of inverting suburothelial purse-string suture for closure of cystotomy. The pyramidalis and rectus abdominis muscles are being retracted from midline. The upper part of the diagram is toward the pubis. (*p*) Peritoneal reflection; (*s*) stay-sutures through cystotomy.

FIG. 5. Application of Z-suture, over completed purse-string suture for reinforced closure of cystotomy. (*p*) Peritoneal reflection.

FIG. 6. Anterior view of lower abdomen and genital region. (*a*) Transverse suprapubic incision through skin crease about 2 cm. cephalad of cephalic ramus of pubic bone and about 10 cm. long; (*b*) midline counterincision, about 2 cm. long, through skin and subcutaneous fatty tissue; outlet for prevesical drain.

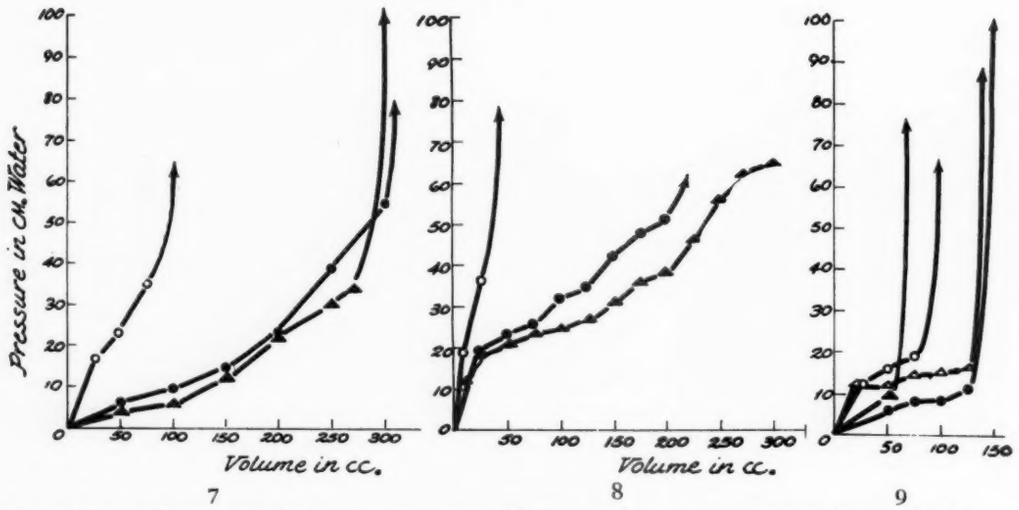

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FIG. 7. Pre- and postoperative cystometrograms (VAH 34450). –▲–▲– preoperative, on day of surgery; –o–o– third postoperative day; –●–●– thirty-eighth postoperative day.

FIG. 8. Pre- and postoperative cystometrograms (VAH32973). –▲–▲– preoperative, on day of surgery; –o–o– first postoperative day; –●–●– fourth postoperative day.

FIG. 9. Pre- and postoperative cystometrograms (VAH 32605). –▲–▲– preoperative, on day of surgery; –o–o– first postoperative day; –●–●– fifth postoperative day; –△–△– fifteenth postoperative day.

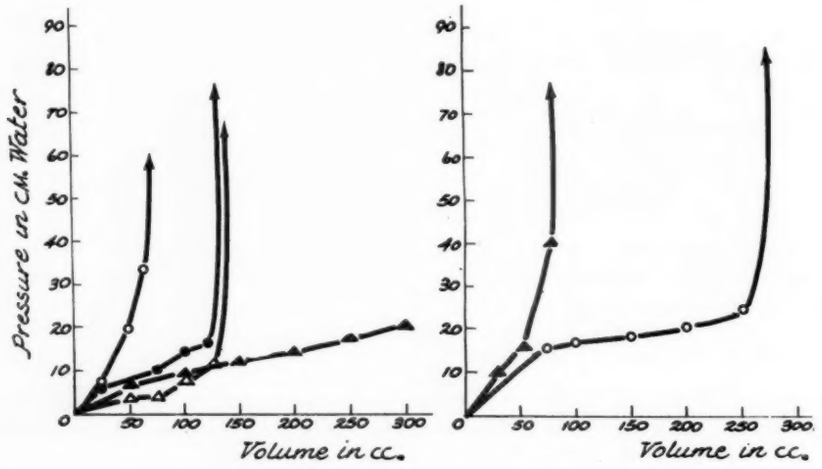

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FIG. 10. Pre- and postoperative cystometrograms (VAH 31922). –▲–▲– preoperative, on day of surgery; –o–o– first postoperative day; –●–●– third postoperative day; –△–△– sixth postoperative day.

FIG. 11. Cystometrograms (VAH 31062). –▲–▲– fourth postoperative day; –o–o– fifteenth postoperative day.

patient (VAH 39891), who had intermittent complete urinary obstruction, was febrile preoperatively in spite of sterile urine and negative urinalysis. His fever was refractory to all antibiotics. Prostatectomy was performed under intravenous oxytetracycline drip. He became definitely afebrile on the fifth postoperative day. There were abscesses in the adenoma.

Vesical spasms occurred in two cases (VAH 32973 and 35632) but were few to absent by the second postoperative day. We have avoided using parasympathomimetic blocking drugs such as atropine and banthine.®

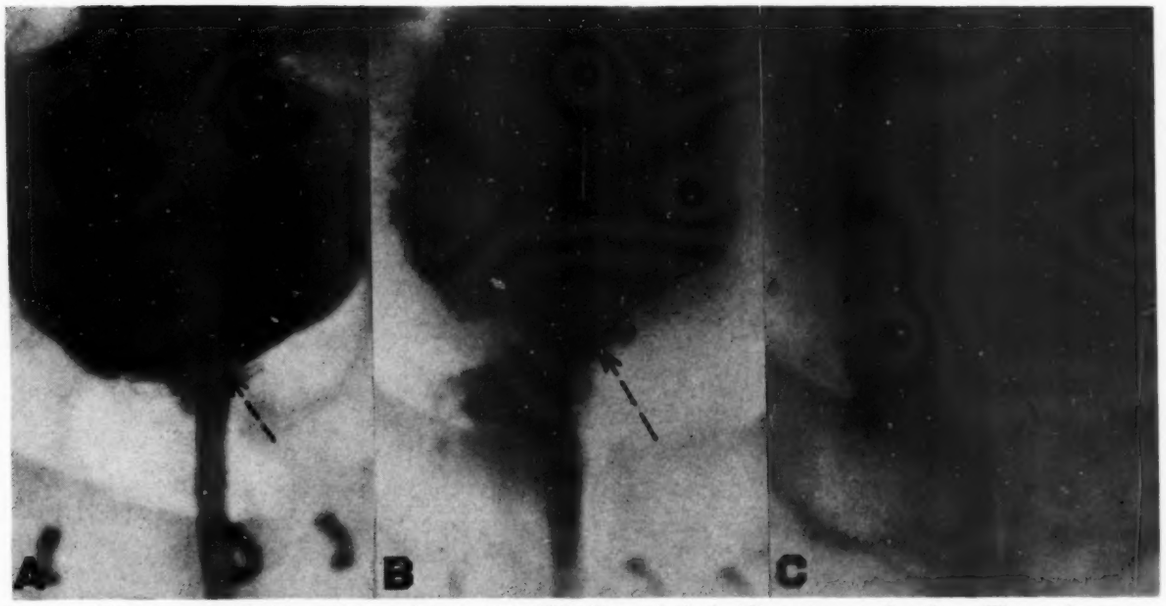

FIG. 12. Cystograms (VAH 31062) on fourth (A), fifth (B) and sixth (C) postoperative days. The catheter was definitively removed on the second postoperative day and was temporarily reintroduced only for the cystogram. Note widening of the internal urethral orifice (arrow) between fifth and sixth postoperative day, paralleling disruption of the three transverse sutures confirmed by cystourethroscopy.

Other postoperative complications were: reaction to penicillin or streptomycin (probably the former) with sloughing of practically the entire cuticle (VAH 27560) but without severe systemic reaction; two cases (VAH 28924 and 33634) of epididymitis; and one case (VAH 32299) of a draining subcutaneous purulent "stitch abscess" which proved to be an extruding 4 by 8 inch gauze sponge readily removed through the suprapubic prevesical drainage site several months after operation. The sinus then healed rapidly. In one case (VAH 42674) thrombophlebitis developed.

Postoperative vesical tonus and pollakiuria. On the first postoperative day, after removal of the catheter, the bladder was capable of holding only 30 to 90 cc. of urine and the patient had to void every thirty to ninety minutes. Pollakiuria was sometimes tantamount to incontinence and was due to markedly increased vesical tone. (Figs. 7 to 11.) One patient (VAH 30166) had a hypotonic bladder preoperatively which reversed to hypertonic immediately after operation. (Fig. 10.) Thus a preoperative hypotonic bladder emptied completely on the first postoperative day and on succeeding days. One patient (VAH 32605) had a markedly hypertonic bladder with small capacity preoperatively; this gradually augmented in capacity after prostatectomy. (Fig. 9.)

Vesical hypertonicity gradually diminished to normal postoperatively. (Figs. 7 to 11.) When patients were ready for discharge on the eighth postoperative day, they were emptying their bladders in volumes usually exceeding 200 cc.

Fate of hemostatic sutures. Daily postoperative cystograms (Fig. 12) and cystourethroscopy in several good-risk cases (VAH 27465, 31394, 31922, 32605, 33473, 31062 and 34450) showed that the No. 1 plain catgut transverse sutures across the prostatic fossa became disrupted by partial absorption between the fifth and seventh postoperative days, confirming the observations of Hryntschak.[32,33] No gross incrustations occurred.

Fate of the prostatic fossa. It is commonly observed that the prostatic fossa contracts rapidly on the operating table. Further shrinkage is evident postoperatively in cystourethrograms. There has been no stenosis of the internal urethral orifice. Two patients (VAH 39891 and 27041) who had pyuria and difficulty in voiding two months postoperatively were found by urethroscopy to have an epithelized narrow fibrous band across the distal end of

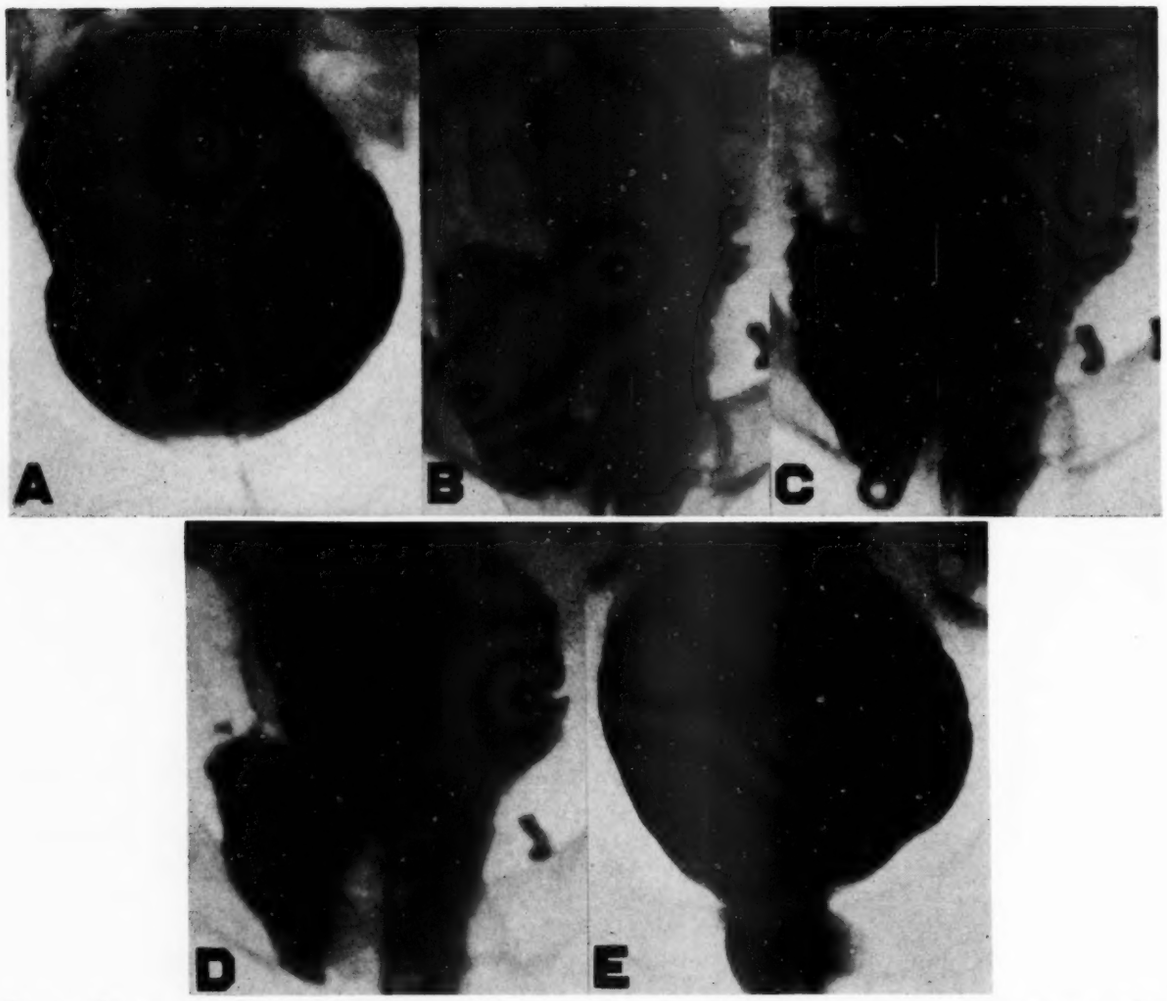

FIG. 13. Cystograms of patient (VAH 34450) whose capsule was perforated during surgery. A, preoperative film; (B), third postoperative day; C, fourth postoperative day; D, fifth postoperative day; E, sixth postoperative day. Safety pin seen in B and C was attached to the prevesical Penrose drain.

the prostatic urethra. This was resected transurethrally.

Another patient (VAH 31973), who was relieved of an 80 gm. adenoma and who underwent suture hemostasis together with two transverse sutures across the prostatic fossa, had his bladder drained suprapubically by an indwelling catheter for four days. The urethral catheter was removed on the seventeenth postoperative day, at which time the patient voided freely and was dry at the incision. At this writing, over a year later, he continues to have a wide open vesical neck. We are convinced that the plain catgut sutures as such will not produce stenosis, even with delayed closure of bladder.

Fate of the inverted portion of the bladder. Cystoscopy during the third postoperative week disclosed almost complete involution and total epithelization of a slightly wrinkled dimple in a small mound at the dome of the bladder.

Residual urine was invariably absent from time of removal of the catheter.

Force of stream was normal after bladder capacity was largely regained.

Incontinence. There has been no permanent incontinence. Usually continence was complete within two weeks. Two patients (VAH 31375 and 34450) showed delayed return of continence. This was probably due to trauma to the external sphincter during enucleation. Extracapsular extravasation due to traumatic

perforation of capsule occurred in one of the patients (VAH 34450). (Fig. 13.)

Blood loss at surgery was concurrently replaced and was excessive largely because of time taken to explain and perform the hemostatic suturing in a teaching institution. We have put the average at a generous 600 cc., usually measured by weighing the sponges and measuring the volume of fluid in the bottle connected to the aspirator. Only one patient had to have transfusion postoperatively. This case has been discussed under "postoperative hemorrhage." In most cases the hematocrit upon discharge was practically identical with the immediate preoperative hematocrit.

Postoperative blood urea. The blood urea tended to rise a few milligrams during the immediate postoperative period but soon reverted to normal. A uremic patient who had elevated but stable blood urea at the time of prostatectomy showed no rise even on the first postoperative day (VAH 46355).

Immediate postoperative ureteral obstruction. Care was taken not to enclose the ureteral orifices in the sutures. A few good-risk patients underwent intravenous pyelography on the second postoperative day and showed no ureterovesical obstruction. One patient (VAH 32605) showed no such obstruction although three transverse sutures had been applied to his prostatic fossa. In an early random case complete regression of preoperative bilateral hydronephrosis occurred after a Hryntschak adenomectomy.[33] Partial regression of hydronephrosis and hydroureter occurred in a seventy-seven year old man with an adenoma weighing 92 gm. (VAH 34982).

Weight of tissue removed (mostly adenoma) was 5 to 96 gm., averaging 47 gm.

Age of patients ranged between fifty-four and seventy-nine years. The average age was sixty-five years.

Number of transverse sutures averaged 1.4. We now rarely apply more than two such sutures, and usually only one.

Definitive removal of catheter was by average early on the second postoperative day. In our more recent cases the catheter has, to advantage, been removed on the first postoperative day. Port wine–colored urine has been no contraindication to removal of catheter. In the few cases in which there was leakage suprapubically with indwelling catheter during the day of surgery, leakage definitively stopped immediately after removal of the catheter.

Cases with open perineal biopsy. Open perineal biopsy for nodules, which proved to be small foci of hyperplasia, was done in three cases (VAH 25450, 25581 and 36805). The prostatic capsule was re-apposed with interrupted No. 1-0 chromic catgut after the biopsy. The biopsy specimens were carefully studied in fixed preparations. Transvesical adenomectomy was carried out in the usual manner on the eighth or ninth day after biopsy. No difficulty in enucleation and no leakage through perineal incision occurred.

Perforation of the prostatic capsule occurred inadvertently at least once (VAH 34450). The catheter was removed from this patient seventeen hours postoperatively; he remained dry at incision. His postoperative course was smooth although daily cystograms (Fig. 8) and cystometrograms were performed in the immediate postoperative period. The prevesical drain was removed on the fifth postoperative day. The transverse suture was disrupted by partial absorption on the fifth postoperative day. No diverticulum resulted from this perforation. (Fig. 13E).

Poor-risk patients (VAH 27230, 23095, 28391, 32299, 33305 and 46751) had arteriosclerotic heart disease (ASHD) with decompensation and were regulated with daily digitoxin. One patient (VAH 23095) also had advanced portal cirrhosis and diabetes mellitus; he died one year postoperatively in hepatic coma. Another patient (VAH 33305) had had myocardial infarction three months previously; preoperative medication was 100 mg. nembutal,® 8 mg. morphine sulfate and 0.4 mg. atropine sulfate hypodermically; anesthesia was by ethylene-ether induction with ether maintenance. Our goal was to attain optimal cardiovascular-pulmonary and metabolic states in every patient prior to adenomectomy.

For this series it was decided to treat poor-risk patients with open adenomectomy rather than transurethral resection, mainly for two reasons: (1) We had adequate suture hemostasis. (2) Our residents were accumulating quantitative data on operative weight gain of patients undergoing transurethral resection of the prostate. They found that the amount of fluid absorbed during transurethral resection varies directly with blood loss (about 9 cc. of fluid absorbed per 1 cc. of blood lost) and that

absorption of as much as 1,200 cc. within one hour of resection was not uncommon.

Emergency adenomectomy. Three patients (VAH 31062, 42674 and 47581) underwent emergency transvesical adenomectomy for postcystoscopic hemorrhage. Approximately 500 cc. of blood were evacuated from each bladder. In each case the prostate was still bleeding profusely when the bladder was opened. The postoperative course was uneventful; the catheter was removed on the first to second postoperative day and one patient (VAH 30162) was catheterized shortly after operation for daily cystograms and cystometrograms. (Fig. 11.) Morbidity, sepsis from repeat irrigation and unnecessary blood loss may be prevented by emergency enucleation if the patient receives adequate transfusion immediately before anesthesia and surgery. Since postcystoscopic hemorrhage is always imminent, although uncommon, we advise cystoscopy only after the patient has had optimal correction of his cardiovascular-pulmonary and metabolic states, i.e., after he has been evaluated and regulated. This was so in these three cases.

Multiple simultaneous surgery. One patient (VAH 33473) underwent ureterotomy through an anterior muscle-splitting incision for removal of a large, completely obstructing calculus in the lumbar portion of the ureter. This was immediately followed with transvesical adenomectomy. Trauma of a repeat anesthesia and "alarm reaction" were thus avoided.

Contraindication to transvesical adenomectomy is a defect in the lower central abdominal wall. Inguinal hernia, repaired or small, has not proved to be a contraindication. Several of our patients had undergone inguinal herniorrhaphy and some had unrepaired inguinal hernias which actually regressed after suprapubic prostatectomy. One poor-risk patient (VAH 33305) had two repaired inguinal hernias which at the present writing, over one year after prostatectomy, show no sign of recurrence.

COMMENTS

Hemostatic suturing at prostatic fossa. Tamponade "hemostasis" at the prostatic fossa, by pack or inflated balloon, is being viewed with increasing disfavor because of unreliability and tendency for late sequela such as sepsis or hemorrhage. This also applies to hemostasis by traction on an indwelling balloon catheter. It is best not to interfere with contraction of the prostatic fossa, which aids venous hemostasis. The most serious bleeding after enucleation occurs from fine arteries at the margins of the prostatic fossa: prostatic branches of the inferior vesical artery or arteries. This was known to Walker,[60] Mertens,[41] Lower,[38] Harris (*loc. cit.*) and others who have applied hemostatic suture ligatures to the margins of the prostatic fossa, and was thoroughly supported by the detailed studies of Flocks[11] on the arterial supply to the prostate. Minute spurters from pudendal and middle hemorrhoidal arteries to apex and lateral margins of the prostate, respectively, can be controlled by a few minutes of pressure on a gauze sponge in the prostatic cavity. Harris originated the deep transverse hemostatic suture through the prostatic cavity. He ligated this suture before introducing the urethral catheter, which was often difficult. Hryntschak[32,33] introduced the catheter before ligating this suture and included the catheter within it. This suture packs venous ooze within prostatic fossa and permits natural contraction of fossa.

Suture hemostasis at the prostatic fossa was first systematized by Harris (*loc. cit.*). His technic was practiced often by Hryntschak,[25–27] who found it a significant advance. Harris' method was simplified and improved by Hryntschak.[32,33] Sorrentino[54] used Hryntschak's modification in 508 cases, with over-all satisfaction.

Among those who have closed the bladder primarily, Hey,[24] Reinle and MacDonald,[52] Stock and Wells,[55] Ritter and Ritter,[53] Anda[1] and Møllerud[45] have fulgurated bleeders after transvesical enucleation. Hemostasis by suture is more secure than by fulguration, hence the practical absence of secondary (i.e., delayed) hemorrhage after transvesical adenomectomy with suture hemostasis. The precariousness of hemostasis by fulguration is recognized by surgeons experienced in transurethral resectino (O'Brien et al.[48]). Some authors who have closed the bladder primarily have used cellulosic acid (oxidized cellulose, oxycel®) in the prostatic fossa for hemostasis (Stockwell,[56] Eikner,[10] Reinle and MacDonald,[52] Stump and Thumann,[57,58] Borgström,[5] and Møllerud[45]). This substance produces an artificial clot with marked affinity for erythrocytes. Its disadvantages, especially in a primarily closed bladder, are: (1) obstructive, essentially nonabsorbable particles in the bladder and pros-

tatic fossa which can hinder micturition; (2) inhibition of thrombin; (3) inhibition of epithelization; and (4) existence of a nidus in which bacteria can multiply. Some of the aforementioned authors also have used gelfoam,® which is less objectionable than cellulosic acid.

Primary purse-string closure of bladder. Primary closure of the bladder after transvesical adenomectomy has been sporadic (Grunert,[17] Mertens,[41] Lower,[38] Hey,[24] Reinle and MacDonald,[52] Stockwell,[56] Eikner,[10] Stock and Wells,[55] Stump and Thumann,[57,58] Bøgrgström,[5] Anda,[1] Chimenti and Saraceni,[9] Ritter and Ritter,[53] and others). Primary closure of the bladder is warranted when there is adequate hemostasis at the prostatic fossa.

Closure of the cystotomy by an inverting suburothelial purse-string suture was apparently first performed on patients by Hryntschak.[32] Brenner[8] studied bladder closure in dogs and human cadavers. In one dog he actually inverted the already everted muscularis by using an inverting purse-string suture. He pointed out that the earlier contemporary protagonists of suprapubic cystotomy, i.e., as against perineal cystotomy, were proponents of bladder closure. Sepsis, resulting from lack of prevesical drainage and inadequate bladder suture, then discouraged primary closure of the bladder. By distending the bladder in human cadavers Brenner showed the mechanical advantage of the purse-string suture compared to longitudinal suturing. His priority in the use of purse-string sutures for bladder closure (in dogs and human corpses) was recognized by von Hacker[18] who suggested closure with purse-string sutures of silk in series. Prives[51] studied various types of bladder closure with silk and catgut. He presented a detailed summary of all previous work.

Early catheter-free micturition. It is noted herein that a probable advantage of early postoperative catheter-free micturition without leakage is prevention of stenosis at the vesical neck. Another advantage is freedom from a major avenue of sepsis. The catheter is a foreign body forming contact with a raw wound—the prostatic fossa—and communicating with a bacteria-rich exterior. If a patient could empty his bladder completely, as did all of our fifty patients, a catheter would be undesirable. In analysis of febrile responses following transurethral resection Marshall[40] noted that ability to remove the catheter early was favorable. In our series the average maximal oral temperature was 100.6°F. The average postoperative day of maximal temperature was 1.4, and the average postoperative day when the patient remained afebrile was 3.7. Our patients were free from sepsis even when the bladder was infected preoperatively.

Another disadvantage of the indwelling catheter is that it tends to reduce ambulation; the elderly patient feels anchored to bed. On the other hand, when the capacity of his bladder is temporarily reduced by increased myovesical tone, he is apt to get out of bed frequently to void. A further disadvantage of the indwelling catheter is sporadic pain referred to penis during vesical contraction.

Blood loss. The average amount of blood lost at surgery was at most 600 cc. This relatively large loss was mainly due to time lapse while residents were taught hemostatic suturing. The blood lost was almost concomitantly replaced on the operating table. An important finding was absence of necessity for postoperative transfusion except in one case in which there was a lapse in operative technic. Hryntschak and Kanzler[34] reported an average loss of 258 cc., measured by the method of Gatch and Little.[13] Goodyear and Beard,[16] and Headstream and Marrin[23] found that a distended "hemostatic" bag in the prostatic fossa increases blood loss, to say nothing of pain and spasm. Their blood loss did not seem to be related to hyperpiesia or age. In five cases of transvesical enucleation by Goldstein and Rubin[15] the average operative blood loss was 278 cc. Pilcher and Sheard,[50] Nesbit and Conger,[47] and Goodyear and Beard[16] have measured blood loss during transurethral resection. Nesbit and Conger reported that in 22 per cent of 100 consecutive cases over 250 cc. were lost and that postoperative bleeding was probably twice as much.

The patient who has undergone transvesical enucleation and who has had suture hemostasis at the prostatic fossa will be practically free from secondary hemorrhage. Harris did not experience it in over 350 cases; Hryntschak[33] had only one in 300 cases; we have had one in 50 cases. Our patient, however, who had abnormally low prothrombin activity, had only a light hemorrhage which never caused obstruction. Transvesical enucleation with "hemostasis" exclusively by gauze pack or balloon

catheter can result in severe secondary hemorrhage during the period of hospitalization in over 5 per cent of the cases.[61]

Fate of the prostatic fossa. It is common experience that the prostatic fossa begins to shrink soon after enucleation. Goodyear and Beard[16] note that the prostatic fossa contracts 50 per cent within a few minutes after enucleation. Within the first six to twelve postoperative hours the fossa contracts by another 25 per cent; it then contracts gradually until completion.[16]

Stenosis at the vesical neck, complete or partial, has been noted after all methods of open prostatectomy. Millin[43] found an incidence of 2 per cent even in the "best of hands." When stenosis occurs, it begins and is complete within a few to several weeks postoperatively (Walker,[60] Irwin,[36] Millin,[43] Goldstein and Rubin,[14] Brea,[7] Nelson and McCormack,[46] Bitschai and Axler,[4] Mimpriss[44] and others). There has been no stenosis in our series and none in the 300 cases of Hryntschak.[33] Hryntschak[25–27] and Harris[19–22] reported no stenosis in a total of 434 cases of adenomectomy using the unmodified method of Harris (371 from Harris and 63 from Hryntschak), in which the bladder was closed primarily. As noted heretofore, the transverse sutures of plain catgut are disrupted by partial absorption between the fifth and seventh postoperative day.

In the stenosing cases of Goldstein and Rubin,[14] Walker[60] and Irwin, and most of those of Millin,[43] there was suprapubic urinary drainage. In some of the cases of Millin[43] and Mimpriss[44] the patients had had enucleation retropubically or perineally and urine drained through the incised prostatic capsule.

Postoperative stenosis at the vesical neck has been divided into membranous and cicatrization types by Millin.[43] The former was ascribed to cross union of torn edges of vesical urothelium over the prostatic cavity and was considered to occur only if micturition had been delayed until the membrane had formed. The latter was ascribed to operative deactivation of the trigonal muscle fibers, thus permitting the circular muscles at the vesical neck to close the vesical outlet unopposed by pull of the trigonal muscle. This was thought to result in fibrosis and shelf-formation at the vesical outlet. This hypothesis is supported by the anatomic studies of Hunter[35] and Uhlenhuth[59] on the musculature of the bladder. Such stenoses can usually be relieved by "revision" of the vesical neck through cystotomy, as the aforementioned authors and Nelson and McCormack[46] testify.

Prevention of stenosis may be aided by removing a generous wedge from the posterior lip of the bladder after the enucleation (Millin), although stenosis has occurred even after this had been done (Bitschai and Axler[4]). Harris did not excise a wedge from the posterior lip of the internal urethral orifice but sutured this lip down to the prostatic capsule in the midline. The most reliable prophylaxis appears to be precise primary closure of the bladder with early postoperative catheter-free micturition. Presumably, micturition maintains the vesical neck open. This appears to be the cogent explanation for absence of such stenosis with adequate primary closure of bladder.

Preoperative vasectomy was not performed routinely. There were two cases of epididymitis. We have the "clinical impression" that if vasectomy is to be of value in reducing incidence of epididymitis it should be performed before the patient is catheterized or cystoscoped.

Potentia coeundi. The method of transvesical enucleation of the prostate does not damage the sympathetic and parasympathetic nerve fibers of erection and ejaculation because the procedure is performed within the surgical capsule and there is no zone of thermal injury beyond the plane of enucleation. In 291 cases of transvesical enucleation with bilateral vasectomy on patients of an average age of sixty-nine years, the operation did not reduce the ability for intercourse in the vast majority of those who had been preoperatively capable.[12] Often there was a rise in libido. One hundred unselected patients who had undergone prostatectomy (average age sixty-nine) were interrogated one year postoperatively (Meuser and Haschek, 1952). Up to the age of seventy there was no significant change in the potentia coeundi (libido, erection, ejaculation). Above seventy years of age there was a significant drop and no improvement. On the other hand, perineal enucleation of the prostate regularly abolishes capacity for erection and ejaculation.[37] This is believed due to injury to branches of the nervus pudendus and to severing of the nervus perinei. Perineal enucleation, therefore, should not be performed in patients who have potentia coeundi and for whom the latter is important.

Hryntschak's procedure in cases of irreversible uremia. Harris[21] insisted on a satisfactory excretion of indigo carmine (maximal concentration in the urine at ten minutes if given intravenously); otherwise he required preliminary drainage by indwelling catheter. Hryntschak[30–33] required a non-protein nitrogen of 50 mg. per 100 cc. or less. He was successful in attaining that value in practically all cases by employing an indwelling urethral or suprapubic catheter. We are in accord with the generally accepted principle that when the patient is even only slightly uremic, an indwelling urethral or suprapubic catheter should be used until uremia is abolished or until the concentration of blood urea, although elevated, has remained stable for a few weeks. During that critical time the patient's water content and electrolyte pattern should be kept as close to normal as possible.[32]

Hryntschak's method has been used without preliminary delay in two patients with bilateral hydronephrosis and hydroureter secondary to prostatic enlargement. Both patients (VAH 34982 and the patient represented by Figures A and B in the translator's preface to Hryntschak's book[33]) had regression of hydronephrosis and hydroureter. In the writer's experience, histologic preparation of such ureters stained with Masson's trichrome to differentiate connective tissue (blue) from muscle (red) show that these ureters are not fibrosed but have an enlarged muscular coat even in cases of terminal uremia from urinary retention secondary to prostatic hyperplasia. Such ureters would thus be expected to undergo reduction in caliber upon decompression.

Perforation of the prostatic capsule occurred in at least one case (VAH 34450). Use of the prevesical soft rubber drain rendered a benign postoperative course. (Fig. 13.) The catheter was definitively removed on the first postoperative day.

SUMMARY

1. Hryntschak's modification of Harris' adenomectomy was performed in sixty-five unselected cases.

2. The catheter was definitively removed on the first postoperative day in most cases. This appeared advantageous in reducing or preventing morbidity (pain, fever, voluntary confinement to bed). The patients remained dry suprapubically.

3. At that time the patient was able to empty his bladder completely although the initial postoperative tidal volumes were small (30 to 90 cc.), probably because of surgically induced hyper-irritability of the bladder. The patient therefore voided frequently. Few patients had vesical spasm during the first twenty-four to forty-eight hours postoperatively.

4. Tidal volumes increased rapidly after surgery. By the eighth postoperative day they were close to 200 cc.

5. There was one case of postoperative hemorrhage. This was secondary to a lapse in fundamental hemostatic technic.

6. The transverse sutures of plain catgut at the vesical neck became disrupted by partial absorption on the fifth or sixth postoperative day.

7. The Hryntschak procedure was safe even with bilateral hydronephrosis and hydroureter secondary to prostatic hyperplasia. It was safe in prostatic adenoma with abscesses and fever, and in moderate "irreversible" uremia.

8. The Pfannenstiel incision was performed in nearly all cases. A small midline counter-incision at right angle to the main incision was suitable in averting rapid superficial closure of the wound and thus in preventing collection of seropurulent fluid subcutaneously.

9. There was no operative death. There was one postoperative fatality, probably from intracranial hemorrhage in a poor risk patient. Otherwise there was no severe complication. There was no permanent or prolonged incontinence.

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Rupture-Perforation of the Esophagus

A REPORT OF SEVEN CASES

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FOR many years perforation and spontaneous rupture of the esophagus have been known to exist, but it would seem that the clinical picture resulting from these conditions is not widely recognized and as a result the diagnosis is frequently not considered. The seven cases reviewed herein were seen at the Milwaukee County Hospital during the years 1949 to 1954. (Table 1.)

Perforations following trauma, i.e., instrumentation and ingestion of foreign bodies, should be easily recognized and surgical correction offers excellent chances of recovery. Perforations due to intrinsic diseases, those due to spontaneous rupture and those due to external trauma are more difficult even for the experienced observer and temporizing is the rule. Eliason and Welty,[2] in a review of the literature in 1946, listed forty-one cases of spontaneous rupture with an associated mortality of 100 per cent. In more recent years, however, additional cases have been reported with reduced mortality. Mackler,[6] in 1952, cited sixteen cases treated by thoracotomy and primary suture. Of these, eleven survived giving a mortality of 31 per cent.

The causes of perforation of the esophagus may be divided into extrinsic, intrinsic and spontaneous rupture. Extrinsic causes are instrumentation, swallowing of foreign bodies, penetration of extrinsic infections, aortic aneurysms and external trauma. Intrinsic causes are tumors of the esophagus, esophagitis, ulceration and diverticula.

The first case of spontaneous rupture was that reported by Borhaave in 1724. Ridgeway and Duncan[9] listed thirty-five cases of spontaneous rupture. Of these, eighteen had obvious alcoholic histories. Numerous references have been made to gluttony, alcoholic debauch, forceful regurgitation and rupture.

McKenzie[7] reported that the weakest point of the esophagus is just above the diaphragm. He believed the left lateral wall was more prone to rupture. This work has been confirmed in animals by Mackler.[6] These findings have been borne out in both our operated and autopsied cases. Williams and Boyd,[10] as a result of animal experiments, postulated that rupture must be preceded by some weakness of the esophageal wall. We believe that rupture can and does occur without any evidence of preexisting disease.

Mackler[6] has postulated that spontaneous rupture occurs as a result of severe vomiting. In instances of repeated vomiting or in the presence of alcoholic intoxication there may be a lack of coordination of the vomiting centers. Physiologically, vomiting results from an increase of intra-abdominal pressure due chiefly to sudden contracture and descent of the diaphragm and contracture of the abdominal muscles. An incisure appears at the lower end of the stomach and the cardia opens. The stomach contents are thus pushed up into the esophagus. If, however, the structures at the upper end of the esophagus, namely, the cricopharyngeus muscle and glottis, do not open simultaneously, there is great increase in intraesophageal pressure. This results in potential rupture at its weakest point, namely, the lower third, just above the cardioesophageal junction.

Some patients may have esophagitis or esophageal ulcers prior to perforation. This may have been the case in some of our mental patients and in others too ill to give an adequate history. The diagnosis of esophageal ulceration is notoriously difficult to demonstrate by fluoroscopy.

Cases of rupture due to external trauma are reported by Aldrich and Anspach,[1] Hunt[3] and Pembleton.[8] One of our cases falls into this group. Under these circumstances rupture is probably facilitated by sudden trauma to the

TABLE I
SUMMARY OF CASES

Case	Age and Sex	Duration	Emphysema	Vomiting	Results	Surgical Treatment
I	74,M	48 hr.	Yes	Yes	Died in 22 days	Closed drainage
II	68,M	5 hr.	No	Yes	Died in 34 hr.	None
III	65,M	?	Yes	?	Died in 36 hr.	Repeated thoracentesis
IV	56,M	4 days	Yes	Yes	Died in 19 days	Closed drainage and thoracotomy
V	26,M	2 hr.	Yes	No	Died in 20 hr.	None
VI	57,M	6 hr.	Yes	Yes	Died in 7 days from enterocolitis	Thoracotomy
VII	53,M	72 hr.	No	Yes	Recovered	Thoracotomy

abdominal muscles with increase in intra-abdominal pressure similar to that which occurs in vomiting.

Several cases in which rupture occurred during an epileptic attack have been reported in the literature.[5] These ruptures were noted shortly after a heavy meal had been eaten. Here, undoubtedly, the mechanism is similar.

Pathology. In spontaneous cases the pathologic picture usually consists of a linear tear in the left lateral portion of the esophagus just above the diaphragm. The margins of the rupture show round cell infiltration of mucosa and muscularis with some destruction of the epithelium. In most cases microscopic evaluation is difficult because of adjacent digestion and infection. Rupture occurs first into the mediastinum. Usually in a few hours digestion of the mediastinal pleura occurs and it ruptures into the left and sometimes both pleural cavities. Air first dissects up the mediastinum, then into the root of the neck, and finally over the anterior chest. Subcutaneous emphysema occurred in five of our seven cases. It was not present in Case VII because immediate perforation occurred into the pleural cavity, offering free exit for the fluid and air and thereby resulting in a lack of dissection into the upper mediastinum. Prior to extension of the emphysema in the subcutaneous tissues or rupture into the pleura with resulting pneumothorax, a diagnosis can only be suspected by the presence of mediastinal air or fluid level in the upright chest film.

The cases of spontaneous ruptures and most of the perforations of esophageal ulcers, if allowed to proceed unaltered, invariably result in death from overwhelming toxemia due to extensive mediastinitis and irritative gastric contents in the pleural cavity.

Signs and Symptoms. The clinical picture is usually fairly typical. Some patients give a history of having been on an alcoholic spree with resultant severe vomiting. Most patients give a prior history of upper gastrointestinal distress or vomiting, or both. Following this they are seized with a severe pain in the chest beneath the sternum or epigastrium with radiation into the back. The pain is excruciating and constant, and the taking of food or water only accentuates it. Occasionally, the pain will make its appearance after a blow to the abdomen or chest. If seen within the first few hours (as in perforated peptic ulcers), the picture is fairly clear. If seen after several days, pain may not be prominent and signs of toxicity are predominant.

Examination will reveal a patient who is acutely ill with fever, rapid pulse and sometimes, shock. The respirations are rapid and labored, especially with pleural extension, and the skin is cold and clammy. The patient may appear cyanotic, and the upper abdomen may be tender and rigid. These may be the only findings in the early stages. Roentgenograms of the chest, if taken early, usually show mediastinal emphysema. If taken later, however, more characteristic signs become evident.

The chest shows signs of pleural effusion unilaterally or bilaterally. Unilateral involvement is usually confined to the left chest. The heart and trachea may be shifted to the right. In early stages the history and onset may suggest a perforated peptic ulcer. Several instances have been reported in which surgeons performed abdominal explorations for ruptures of the esophagus. The initial impression in our last two cases was that of perforated peptic ulcer. Subcutaneous emphysema of the neck is a prominent sign and occurred in seventeen of twenty-seven cases of Ridgeway and Duncan,[9] and in 50 per cent of the cases reported by Eliason and Welty.[2] This sign was present in five of our seven cases. A sign which has been of some value to us is the presence of a crackling sound synchronous with the heart beat. This was present in two of our last three cases.

X-ray examination at later stages shows mediastinal emphysema, pneumothorax, pleural effusion or pneumohydrothorax. On suspicion of a perforation the diagnosis can be readily established by having the patient ingest 40 to 60 cc. of radio-opaque oil under fluoroscopy. The media can be readily seen to enter the pleural cavity or a sinus tract into the mediastinum. Barium is somewhat irritative and therefore oil is preferred.

Several other conditions may be confused initially. These consist of coronary occlusion, acute hemorrhagic pancreatitis, spontaneous pneumothorax, spontaneous mediastinal emphysema, dissecting aneurysm of the aorta, rupture of the diaphragm and incarcerated hiatal hernia. The previously described x-ray examinations should serve to separate these conditions from that of perforated esophagus.

Treatment. Although some of these patients may be in terminal states when first seen, early surgery offers the only hope. Treatment with antibiotics has not influenced the course of the disease in our experience. Some cases have been treated by drainage of the chest. This was attempted in three of our earlier cases with disappointing results.

The preferred method of treatment consists of thoracotomy and primary suture of the esophagus. Water-seal drainage of the chest is then carried out, and an inlying Levin tube is retained about three days for feeding purposes. Adequate antibiotics and liberal treatment of the shock by blood transfusion has also helped greatly. Four of our patients did not undergo surgery, and all died. Three were treated by thoracotomy and of these, one survived. The first patient was operated upon at a very late date, and the second patient died from an unfortunate complication. The third lived, but had a very stormy and extended convalescence.

The time factor is of great importance, as it is in perforations of the stomach and duodenum. The patient may die within a few hours. On the other hand, in some cases surgery may involve dealing with an exceedingly ill individual who is poorly able to tolerate the effects of major surgical intervention. In our patients operated upon, however, it is remarkable how much improvement occurred upon opening the mediastinum, closing the esophagus and cleaning out the pleural cavity. After several days the torn esophagus becomes exceedingly friable and difficult to close, as illustrated by the disruption of the suture line in two of our cases in which late surgery was performed.

CASE REPORTS

CASE 1. O. L., a seventy-four year old white man, entered the hospital complaining of upper abdominal pain with radiation into the chest and left shoulder. Twenty-four hours previously diarrhea had developed followed by vomiting. He had been in this hospital in 1941 with dyspepsia. Gastroscopic and fluoroscopic examinations made then revealed no significant findings.

On the present admission the temperature was 99.6°F., pulse 106, respirations 36 and blood pressure 139/70. The neck veins were engorged, respirations were labored and subcutaneous emphysema extended upward from the clavicles bilaterally. The breath sounds were decreased throughout the entire right lung with hyper-resonance in the left apex and dullness in the left base. The abdomen was distended, and voluntary rigidity and tenderness were present in the epigastrium and right upper quadrant.

Left thoracentesis removed 550 cc. of brownish material, and resulted in relief of symptoms. Roentgenograms of the chest showed patchy infiltrations of the lower third of the right base and hydropneumothorax on the left.

Daily chest aspirations were instituted and on the tenth day closed drainage was established. Three days later gastric contents were

noted in the drainage tube, Levin suction was begun, and parenteral fluids were started. The patient was confused and uncooperative and surgery was deemed inadvisable. The patient's course continued to go downhill and he died twenty-two days after admission.

Autopsy showed three areas of ulceration of the lower third of the esophagus. The largest measured 1 by 3 cm. and communicated with an empyema of the left chest. Microscopic sections of the perforation showed marked round cell infiltration. There were also two ulcers of the first part of the duodenum. One was attached to the undersurface of the liver and a second was closely attached to the head of the pancreas. The precise pathology of the esophageal perforation could not be accurately determined because of inflammation and reparative changes of three weeks' duration.

It is interesting to note the association of vomiting and pain with the presence of gastroenteritis or peptic ulceration. It is possible to postulate that vomiting may have been an etiologic factor in the calamity. Early operation should have been performed.

Case II. H. M., a sixty-eight year old paretic white man, was admitted to the hospital from a mental institution. He complained of upper abdominal pain of five hours' duration. This was described as being severe and stabbing and occurring chiefly in the epigastrium with radiation under the left costal margin. About one-half hour after the onset of pain the patient began to vomit.

Past history revealed he had noted pain in the right upper quadrant of the abdomen after meals for the past six to seven months. This occurred on the average of once or twice a week. The patient had been in this institution sixteen months previously with homologous serum jaundice.

Examination revealed that the patient was acutely ill. Temperature was 101.4°F. (rectal), pulse 92, respirations 36, and blood pressure 118/36. The respirations were grunting and there were rales in the right base on auscultation and dullness to percussion. The abdomen was protuberant with epigastric tenderness and voluntary rigidity. Bowel sounds were active. The stools were clay-colored, and the patient appeared slightly jaundiced.

There was no subcutaneous emphysema and a Levin tube was not passed. The patient's course was rapidly downhill and he became confused, toxic and cyanotic. The temperature, pulse and respiration increased, and the patient died thirty-four hours after admission.

Autopsy showed 6,000 cc. of brown fluid in the right pleural cavity. The esophagus showed a large ulcerated area in the lower third, approximately 3 cm. in diameter with rolling up of the superior margin. A small perforation, measuring 2 by 3 mm., communicated with the soft tissues surrounding the esophagus and great vessels, and from this point deeply infiltrated the mediastinal soft tissue and dissected along the right main bronchus into the right pleural cavity. Microscopic examination revealed acute necrosis of the wall of the esophagus with surrounding inflammatory reaction. Final diagnosis was acute esophagitis with ulceration and perforation, in addition to cholesterosis of the gallbladder and early biliary cirrhosis.

Case III. R. R., a sixty-five year old white man, was admitted to the hospital in acute respiratory distress and was unable to give any history. He was perspiring profusely, and the lips, hands and feet were reddish blue in color. The patient's landlord said the man had been in good health until one week prior to admission when a mild attack of coryza with cough developed. This was associated with complaints of pain in the low back and left chest. He had been seen by two doctors who told him he had pneumonia. The day of admission, however, the respirations became labored and his landlord called an ambulance.

Physical examination revealed a well nourished white man who was cyanotic and acutely ill. The temperature was 100.4°F., respirations 52, pulse 100, and blood pressure 80/0. Dullness of the lower three-quarters of the left chest and of almost the entire right chest was present. Subcutaneous emphysema was noted on the left side of the neck. Thoracentesis removed 1,400 cc. of mahogany-colored fluid from the left side and 700 cc. from the right. Following this procedure the patient improved. A Levin tube was not passed and during the next twenty-four hours the patient's course continued downhill. Blood pressure dropped to zero, temperature increased to 104° F., and the patient died thirty-six hours after admission. X-rays of the chest prior to death were unsatisfactory because of the patient's inability to cooperate.

At autopsy an ulcer of the peptic type was

found in the lower third of the esophagus. It had perforated into both pleural cavities and the lungs were covered with a thick greenish layer of fibrin. Microscopic sections showed congestion of the vessels with some lymphocytic and plasma cell infiltration. Much of the submucosal tissue appeared pinkish and hyaline. In addition, adenocarcinoma of the transverse colon with metastasis to peritoneum, regional nodes, omentum and pleura was present.

CASE IV. J. P., a fifty-six year old white man, entered the hospital for mental observation and treatment of alcoholism. The patient stated he had had a cold and cough for the past two weeks. Four days previous to admission he awakened feeling very weak and noted a dull ache in the lower abdomen. He induced vomiting by placing his finger in the pharynx. Immediately following this, severe upper abdominal pain radiating through to the back and posterior chest developed. This pain was so severe he could not lie on his back. A doctor was consulted and sent him to a hospital where a chest x-ray was taken. He was then transferred to the County Hospital for mental observation because, "He was so sick and had severe pain but nobody believed him."

Physical examination revealed a man who appeared acutely ill. Temperature was 102.4°F. (rectal), pulse 120, respirations 22, and blood pressure 106/70. Marked subcutaneous emphysema was present on both sides of the neck and extended down over the anterior chest. Dullness of the lower chest and tympany above this with tenderness and guarding of the epigastrium were also noted.

Chest roentgenograms showed pneumothorax with 20 per cent collapse of the left lung. Fluid was present in the left base and air was noted in the mediastinum.

A Levin tube was passed and the patient was placed on parenteral fluids and antibiotics. On the following morning closed drainage was instituted and ingestion of radio-opaque dye revealed a defect of the lower esophagus.

Nine days later left thoracotomy was performed because the patient's course appeared to be going downhill. A 2 by 1 cm. perforation of the lower end of the esophagus was closed with difficulty with interrupted silk sutures. Water-seal drainage was instituted.

Jejunostomy was performed later, but the patient died nineteen days after admission.

At autopsy a defect of the esophagus was found just above the cardioesophageal junction with chronic empyema of the left chest and chronic mediastinitis. Two silk sutures were present but a defect remained. Microscopic examination showed moderate round cell invasion around the perforation with intact squamous epithelium and muscularis up to the margins of the opening. This was classed as an acute perforation, probably spontaneous in nature as no ulcer could be demonstrated.

CASE V. L. B., a twenty-six year old white man, was admitted to the hospital after being struck by an automobile as he was placing flares on a highway. Upon admission he complained of pain in the left lower leg, left chest and sternum.

Blood pressure was 103/70, pulse 116, temperature 98.6°F. He had a compound fracture of the left tibia, and the left chest showed decreased resonance and tubular breath sounds over the base. Tenderness was present over the sternum but there were no fractures. Normal bowel sounds were present and there was splinting and tenderness of the epigastrium.

The next morning a portable x-ray revealed clouding of the left chest. Thoracentesis obtained a small amount of bright blood. By this time the patient had severe pain in the chest and some respiratory embarrassment. The Levin tube was removed and shortly afterward cervical emphysema was noted. The patient's course continued downhill and he died twenty hours after admission.

At autopsy a left pleural effusion containing 2,000 cc. of cloudy grey material was found. There was a linear opening of the right posterolateral surface of the esophagus, measuring 2 by 2.5 cm., just above the diaphragm. This communicated with the left pleural cavity by way of a sinus tract through the pleura. Microscopic examination showed acute necrosis of the edge of this tear with surrounding acute inflammatory reaction.

CASE VI. H. S., a fifty-seven year old white man, entered the hospital complaining of pain in the epigastrium and left back. A duodenal ulcer had been demonstrated and treated previously but symptoms persisted. For several weeks prior to admission he had eaten nothing but soft food. In an attempt to relieve his distress he induced vomiting. Vomiting continued throughout the night, and on the day of admission he was seized with sudden severe pain in the epigastrium and left chest.

Upon admission he was cold and clammy and had considerable respiratory distress. Temperature was 101.8°F. (rectal), pulse 104, respirations 28, and blood pressure 147/88. The chest was clear, and the abdomen was rigid, generally tender and silent. The initial impression was perforated peptic ulcer. However, roentgenograms of the abdomen failed to show free air and chest films showed minimal infiltration in the left lower lung fields. Re-examination an hour later revealed subcutaneous emphysema of the neck tissues. This was followed by chest films after the ingestion of radio-opaque oil. The oil was seen to extend into the pleural cavity from what appeared to be the mid-portion of the esophagus. A diagnosis of rupture of the esophagus was then made and thoracotomy was performed about six hours after perforation. A 2.5 cm. linear opening was found just above the esophageal hiatus in the left anteromedial portion. There was about 250 cc. of brown fluid in the chest cavity with extensive mediastinal emphysema. The esophagus was sutured and a pleural patch oversewn.

The postoperative course was uneventful until the fifth day when severe diarrhea developed. The patient had been on a liquid diet and was progressing well until this point. Chest roentgenograms showed good expansion of both lungs with some residual pneumonia on the left. The diarrhea was thought to be due to staphylococcus enterocolitis, related to aureomycin. This drug was stopped and erythromycin was given, but severe toxemia, confusion, abdominal distention and shock developed and the patient died within forty-eight hours. No autopsy was permitted, and stool specimens for culture were lost. It was believed that death was due to acute staphylococcus enterocolitis.

CASE VII. J. T., a fifty-three year old Negro man, entered the hospital after an acute bout of vomiting and diarrhea. Following the vomiting he experienced sudden severe epigastric and anterior left chest pain. He described this as a tearing sensation. The abdomen was rigid and silent. No emphysema was present. Roentgenograms of the abdomen and chest revealed no free air, but a minimal pneumothorax was noted on the left. He was then transferred to a private hospital where two chest tubes were placed for drainage. These tubes were productive of stomach contents and a defect of the esophagus was suspected. Forty-eight hours later he was returned to the County Hospital mental ward because he became disoriented and uncooperative. The following morning the chest was opened through the seventh left interspace, a 3 cm. opening was sutured and the lower lobe was decorticated. Considerable drainage developed and on the seventh postoperative day an esophageal fistula was demonstrated by x-ray.

On the eighteenth day, after the drainage had decreased markedly, thoracotomy was again performed and the esophagus was successfully closed. The previous drainage site closed and the patient was discharged on the twenty-fifth day.

SUMMARY

Seven cases of perforation of the esophagus are reported. Three of the seven can probably be classified as spontaneous rupture. One was due to perforation of a chronic ulcer, one was due to external trauma, and the other two defy classification. One patient survived. A plea is made for greater awareness of this condition if it is to be diagnosed and treated early. Early surgical intervention with primary closure of the esophagus is urged and would seem to be the only solution to the problem.

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Treatment of Fractures of Shaft of Tibia by Intramedullary Fixation with Lottes' Nail

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THE incidence of unstable oblique and spiral fractures of the shaft of the tibia is considerable; furthermore slow union, delayed union and non-union of these fractures, particularly of the lower third of the shaft, are quite common. Treatment of these difficult fractures of the tibia has not been too satisfactory until the present. Skeletal traction has frequently been associated with delayed union or even non-union of the fracture; there has also been frequent stiffness of the neighboring joints, particularly the subtalar joint. Watson-Jones and Coltart,[1] in a now classic paper on slow union of fractures, showed the severe retardation of union by traction and particularly overpull. (Table 1.) The blood supply of the bone is necessarily disturbed by periosteal stripping at the fracture site incidental to the open reduction. This probably explains, at least partially, the slower union of fractures following open reduction. The incidence of infection is, of course, always greater following open reduction in comparison with closed reduction. It is also frequently difficult and occasionally impossible to close the skin over the shaft of the tibia following the open operative procedure. While satisfactory results followed open reduction and internal fixation by screws or by plates and screws, union seemed also somewhat delayed by the rigid fixation which did not allow approximation of the fragments after absorption at the fracture site. Because of these disadvantages of traction and open reduction of the unstable fractures of the long bones, the principle of medullary fixation of these fractures has been established in recent years as the greater mechanical efficiency of this type of fixation has become evident. Treatment of fractures of the shaft of the femur by intramedullary nails and rods was first accepted; fractures of other long bones did not seem as suitable for this type of fixation. Recently, however, medullary fixation of the radius, ulna and tibia has been worked out and seems to offer great advantage over other treatment methods. Since there is still much opposition to the medullary fixation of fractures,[2] it is worth while to report our experience with a recent small series of fractures of the shaft of the tibia treated by fixation with Lottes' nail.

Description of Nail. This instrument was first described by Otto Lottes of St. Louis in May, 1952.[3–5] It is a tri-flanged nail made of 18-8 S.M.O. stainless steel; it is gently curved, with the curves accentuated at its upper and lower ends. The distal end is pointed, the proximal end is threaded to permit application of a Smith-Petersen introducer. The device is minimally elastic and comes in various lengths (the most frequently used nails are between 11 and 13½ inches long) and in two widths, namely 5/16 and 6/16 of an inch.

Surgical Technic. Following the original technic of Lottes, the patient is anesthetized in the supine position on the fracture table with the hip flexed to 120 degrees and the knee bent to 90 degrees. Traction is applied through the foot plate of the fracture table. A sling under the lower portion of the thigh suspended from an overhead frame serves as counter-traction. Occasionally this traction arrangement has been modified by applying traction to both lower extremities; in transverse or ununited fractures traction is not as important as in unstable oblique and spiral fractures. Very effective traction has been applied in several cases by means of a Kirschner wire through the calcaneus. The fracture of the tibia is then reduced by adequate traction and derotation until the alignment appears clinically satisfactory, the obvious landmark being the outline of the tibial crest. The extremity is draped, leaving a sterile field of the shaft of the tibia. A 1½ inch longitudinal incision is made one fingerbreadth medially to the tibial tubercle, from the middle of the tubercle upward. The distal inch of the incision is carried down to bone. The bone is then windowed

TABLE 1

TIME OF UNION OF 417 FRACTURES OF SHAFTS OF TIBIA AND FIBULA*

Results	Twelfth Week (%)	Sixteenth Week (%)	Twenty-fourth Week (%)	Forty-eighth Week (%)
Closed reduction:				
Continuous immobilization (199)	42	70	95	100
Interrupted immobilization (104)	11	33	74	100
With distraction (16)	..	6	31	97
Open reduction:				
Without infection (46)	9	39	72	100
With infection (52)	2	6	35	91

* From Watson-Jones, R. and Coltart, W. D.[1]

with any suitable instrument, preferably a ⅜ inch brace-type drill, but a small osteotome or gouge can also be used. In my experience a skull trephine is an excellent instrument for this purpose. It is absolutely essential to slant the opening sufficiently to permit introduction of the nail almost parallel to the long axis of the tibia. For this purpose the drill and later the nail are aimed at the crest at the junction of the middle and lower thirds. The exact level at the middle of the tibial tubercle is also important, because lower introduction of the nail will frequently split the anterior cortex of the tibia. The length of the nail is predetermined by measurement of the sound leg from the middle of the tibial tubercle to the tip of the medial malleolus. The nail is then inserted with the solitary flange at the concavity of the nail pointing anteriorly. The nail must be depressed to approximate the long axis of the tibia. It should then be hammered down to just proximal to the fracture site; little resistance should be encountered and the nail should advance steadily. If introduction is difficult, one should immediately withdraw the nail and reapply it accurately as the obstacle must have been due to impingement of the nail against the cortex. A special effort is then made by the surgeon to hold the bone fragments in anatomic alignment while the assistant hammers the nail into the distal fragment. Firm fixation of the fracture is evident as soon as the nail engages the distal fragment. If fixation is not solid, the nail has either slipped to the side of the distal fragment, or has penetrated posteriorly. (Fig. 1.) After some experience this becomes quite obvious; and if transfixion of the fracture is not firm, the nail should be withdrawn at once, the fracture re-reduced and the nail reinserted. As experience accumulates, open reduction of fresh fractures will only rarely be necessary. In Lottes' own series this was only done in three cases of fresh fractures. After the first two weeks open reduction of the fragments necessarily becomes increasingly frequent. Reduction of the fracture and nailing are checked by x-ray examination at the end of the procedure. The incision is closed and a long leg plaster is applied with the knee in a few degrees of flexion. This initial cast is changed after about ten days and a snug walking cast is applied to the mid-thigh with the knee in almost complete extension. In well fixed, suitable fractures the patient is allowed to start full weight-bearing after the application of the walking cast. In very comminuted fractures it is safer to wait for some consolidation of the fragments before weight-bearing is begun. There should be at least one-third to one-half apposition of the cortices of both fragments to start early walking. In the simpler fractures it is almost routinely possible to remove the plaster at the end of two months. My greatest difficulty is to determine functional union. The former tests of clinical union, namely, springiness at the fracture site, pain on straining the fracture site, local tenderness and auscultatory percussion, do not apply to this series because of the solid mechanical fixation and the use of metal. It would be unfair to the patient and, in my opinion, quite unnecessary to withhold weight-bearing without support until trabeculation is demonstrated across the fracture site on x-ray examination. This is a late sign of bony consolidation. At present I am guided by the following signs: (1) bone fragments and nail

FIG. 1. Demonstration of faulty insertion of Lottes' nail.

should have retained the original alignment; (2) there should not be any absorption about the fixing device on x-ray examination; and (3) pain should be absent on a test of weight-bearing without support. It is to be noted that primary callus will be sparse. However, actually it is usually somewhat more marked than in some anatomically aligned fractures fixed with adequate Lane plates and screws and further supported by long leg casts. It is my impression that the fracture line is frequently visible for longer periods of time than in conservatively treated fractures because of this smaller primary callus which fails to obscure the fracture site.

Indications. Medullary nailing of the tibia is only advisable for unstable fractures which would have had to be fixed internally by some other method. In other words, the indications for nailing are essentially unchanged from our previous surgical approach to this problem. Certainly stable fractures of the tibia and particularly fractures of this bone in children should preferably be treated by closed reduction and plaster fixation. Nailing is applicable to almost all fractures of the mid-third of the shaft. In an occasional instance of a very comminuted fracture of the mid-third of the shaft, fixation of the main fragments can only be partially accomplished. This should be recognized and aftercare handled accordingly. All fractures of the upper portion of the lower third of the shaft can be nailed, but the fracture line should not extend to lower than 2½ inches from the ankle. My own experience with one fracture of the upper third of the tibia was not too satisfactory. Due to the wide medullary space in the upper third of the shaft a tilt of the upper fragment occurred, although the fixation was quite solid. This fracture is now healing uneventfully. In view of theoretic considerations and this practical experience, I do not recommend nailing of fractures of the upper third of the tibia, but according to Lottes an occasional fracture of this type can also be nailed. Transverse and short oblique fractures are most suitable, but long oblique and even spiral fractures, segmental fractures and comminuted fractures, even with large butterfly fragments, can be readily nailed.

Results. The Lottes' nail firmly fixes suitable fractures of the tibia. This fixation supplemented by a long leg cast has led to early bony consolidation in all my patients. I have not experienced any cases of delayed union or non-union. These results are due to avoidance of interference of the blood supply of the bone, the lessened danger of infection and the firm fixation. The nail itself does not interfere with the blood supply of the tibia nor are there any documented cases of fat embolisms. Because of the early function the adjacent joints are almost free of any disabling joint stiffness. In a large statistical analysis presented by Lottes, Hill and Key,[4] results are obviously superior to those formerly obtained by open reduction and fixation with plates and screws. (Table II.) Although I realize that their table is evidently prejudiced and their high rate of infection in the plated fractures is way out of line to that tolerated on any good fracture service, the end results of the nailed fractures of the tibia speak for themselves.

Complications. I have not had any patients with bone infections, although eight of twenty cases were open fractures. A superficial skin infection developed in one patient six months after surgery, and subsided promptly after removal of the nail. After open reduction of the fracture in one patient, despite a release incision and the fashioning of a flap, necrosis of the skin developed over the tibial crest. This required skin grafting. The only other serious complication I have encountered was the case of a fifty year old man. Phlebothrombosis with embolization developed eighteen days after fracture and nailing while the patient was ambulatory. Fortunately, he recovered on anticoagulant therapy. Transient fever for

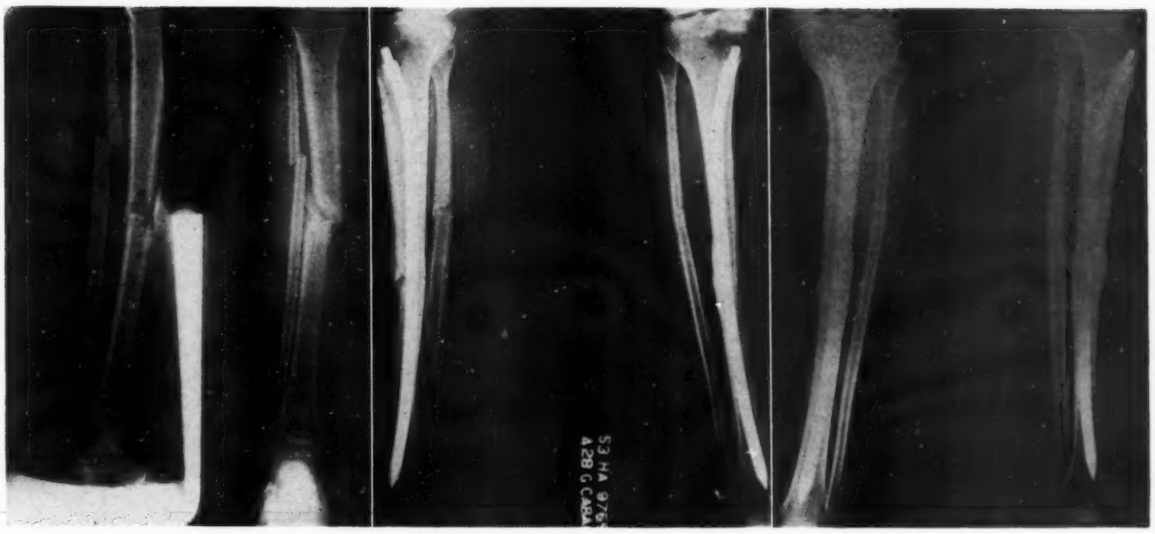

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FIG. 2. Case 1. Open short oblique fracture of mid-third of the tibia with small butterfly fragment. X-ray taken on admission (January 3, 1953).

FIG. 3. Same case. X-ray taken shortly after removal of cast (February 28, 1953).

FIG. 4. Same case. Consolidation of fracture eight and a half months after injury, reduction and nailing (September 18, 1953).

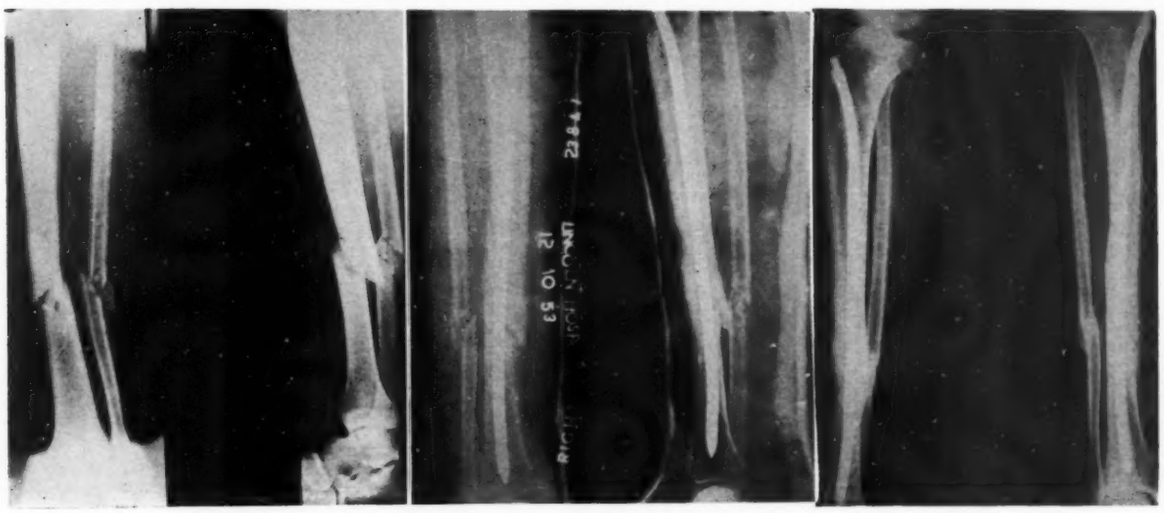

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FIG. 5. Case II. Closed short oblique fracture of the lower mid-third of the tibia and fibula. X-ray taken on admission (August 30, 1953).

FIG. 6. Same case. Walking cast. In retrospect, it would have been safe to have this patient walk without external support at this time (December 10, 1953).

FIG. 7. Same case. The fracture is solidly united. Nail was removed because of prominence of upper end (September 21, 1954).

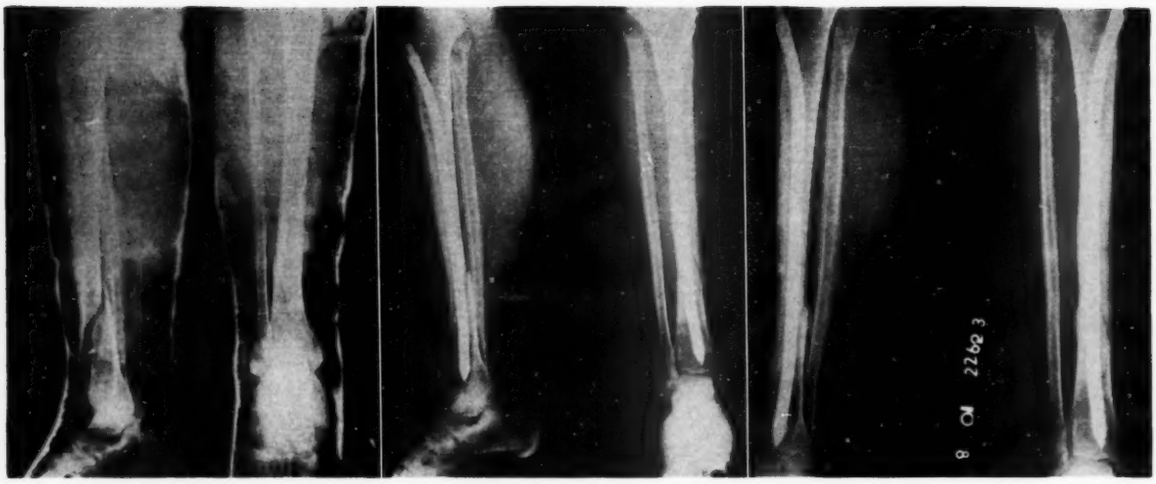

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FIG. 8. Case III. Closed short spiral fracture of the distal third of the tibia in plaster on day of admission (January 11, 1954).

FIG. 9. Same case. One month after removal of cast. Patient walking without support (April 30, 1954).

FIG. 10. Same case. Fracture is consolidated (October 8, 1954).

11 12 13

FIG. 11. Case IV. Closed short oblique fracture of mid-third of tibia in plaster. X-ray taken four days after admission (December 26, 1953).

FIG. 12. Same case. X-ray taken after removal of plaster (April 2, 1954).

FIG. 13. Same case. Advanced consolidation of fracture (September 1, 1954).

TABLE II*

Complications	Closed Reduction		Plate Fixation		Medullary Nailing	
	Simple (%)	Compound (%)	Simple (%)	Compound (%)	Simple (%)	Compound (%)
Non-union	5.6	16.0	13.0	35.0	0	0
Infection, mild	0	16.0	17.3	25.0	1.9	3.3
Infection, severe	0	8.0	8.7	10.0	0	3.3
Loss of skin requiring graft	5.6	12.5	4.3	20.0	0	0
Varus or valgus deformity	9.6	12.5	4.3	0	5.7	13.3
Shortening, more than ¼ inch	19.6	12.5	4.3	0	1.9	3.3
Recurvatum	19.6	8.3	0	0	9.8	6.6
Offset	19.6	8.3	0	0	5.7	10.0
Rotation	2.8	4.1	0	0	0	0
Amputation	0	0	0	10.0	0	0
Thrombophlebitis	0	0	0	0	0	3.3
Number of tibias	36	24	22	20	51	30
Average number of cases of non-union	2	4	3	7		

* From Lottes, J. O., Hill, L. J. and Key, J. A.[1]

two or three days after operation has frequently been observed.

The following brief case reports illustrate the management of fractures of the shaft of the tibia.

CASE REPORTS

CASE I. G. C., a twenty-eight year old woman, was knocked down by an automobile and sustained an open short oblique fracture of the mid-third of the tibia and fibula with a small puncture wound, probably due to perforation of a bony fragment. Closed reduction and Lottes nailing were performed on the day of the accident. The patient was allowed to bear weight one month later. The cast was removed three months after the accident and surgery when union seemed solid. No complications ensued and all joints of the extremity were free. (Figs. 2 to 4.)

CASE II. B. P., a thirty-five year old man, was knocked down by an automobile and sustained a closed short oblique fracture of the mid-third of the tibia and fibula. Initially he was placed in traction. Eleven days after the accident closed reduction of the fracture was attempted. This was not successful and the fracture site had to be opened. The fracture was reduced and a Lottes nail was used for fixation in the usual manner. The patient was allowed to bear weight after a change of cast ten days later. The cast was removed five months after fracture; the patient had an excellent result. Because of prominence of the upper end of the nail, it was removed without difficulty thirteen months after injury. (Figs. 5 to 7.)

CASE III. D. T., a thirty-eight year old woman, fell and sustained a closed short spiral fracture of the distal third of the tibia and fibula. Closed reduction and Lottes nailing were performed two days after the accident and the patient was permitted to bear weight after a change of cast two weeks later. The cast was removed seventy-three days after surgery when union seemed solid. She has not had any further difficulties. (Figs. 8 to 10.)

CASE IV. J. L., a fifty-seven year old man, was knocked down by an automobile and sustained a closed short oblique fracture of the mid-third of the leg. He was initially treated by closed reduction and application of a cast, but alignment was not satisfactory. Thirteen days after injury closed reduction of the fracture was performed and a Lottes nail used for fixation. The patient was allowed to bear weight one month after surgery. The cast was removed three months after injury when the fracture appeared solid. There were no complications. (Figs. 11 to 13.)

COMMENTS

In my opinion, treatment of fractures of the shaft of the tibia by intramedullary fixation with a Lottes nail is a worth while advance in fracture surgery. This method has led to early bony union in fifteen of twenty patients. It is too early to evaluate the results in five patients, but they seem to be convalescing uneventfully. Of course, I anticipate cases of delayed union or non-union, but by and large union seems to be more certain and also accelerated compared to that expected in patients with fractures of the tibia treated by other types of internal fixation. The absence of joint stiffness is most helpful; the early return to function and prompt rehabilitation of the patient is gratifying. The Lottes nail is left *in situ* unless there is any indication for removal, such as local infection or extrusion of the instrument.

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A Study of Certain Factors in the Etiology of Thrombophlebitis in the Lower Extremities Following Surgery

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IT is generally agreed that multiple factors participate in the etiology of thromboembolism following surgery. The apparently inconstant presence of such factors may be due, in part, to the lack of use of known criteria for the diagnosis of peripheral venous thrombosis.[1] In a previous study it was demonstrated that shock and hypotension were present before, during or after surgery in a statistically significant number of patients who later died from pulmonary embolism.[2] It was suggested that shock and hypotension might be etiologic factors in thromboembolic disease.[2] The present study is concerned with an analysis of the possible etiologic role of shock and hypotension in postoperative thrombophlebitis.

In view of the difficulties often encountered in establishing the presence or absence of peripheral venous thrombosis the present study was limited to thrombosis of the veins of the lower extremities, which is relatively easily diagnosed by clinical features such as pain, tenderness and swelling. It was thus necessary to exclude many patients with suspected but not clinically verifiable venous thrombosis occurring in the prostatic veins, the pelvic veins and other areas. Also excluded were patients stated to have thrombophlebitis of one or both lower extremities but on whose case records confirmatory clinical findings had not been noted.

STATISTICAL MATERIAL

The records of ninety-eight patients with postoperative thrombophlebitis involving one or both lower extremities were studied. Particular attention was paid to the occurrence of shock or hypotension before, during or after surgery. These patients had been treated at the Los Angeles County Hospital during the period from 1942 to 1952. In addition, the records of a series of 2,000 major operations of all types were used to provide information regarding the incidence of shock and hypotension before, during and after operation in control patients of approximately the same age as the ninety-eight patients with thrombophlebitis. The criteria of shock and hypotension used in the present study were as follows: (1) shock: a systolic blood pressure of 80 mm. of mercury or less; (2) hypotension: a systolic blood pressure ranging from 81 mm. of mercury to 90 mm. of mercury; (3) relative hypotension: a fall in systolic blood pressure of 70 mm. of mercury or more even though the resulting level remained above 90 mm. of mercury.

STATISTICAL ANALYSIS

The site of thrombosis was stated to be (1) the right lower extremity in twenty-six patients; (2) the left lower extremity in fifty-one patients, and (3) both lower extremities in twenty patients. In one patient definite symptoms and signs of thrombophlebitis were present, but no statement was made as to which lower extremity was involved. Pulmonary embolism occurred in twenty-four patients. Fifty-eight of the patients were women, and forty were men. Their ages ranged from seven to eighty-eight years. Of the fifty-eight women, thirty-six (62 per cent) were below fifty years of age, and twenty-two (38 per cent) were fifty years or older. Of the forty men, twelve (30 per cent) were below fifty years of age, and twenty-eight (70 per cent) were fifty

years or older. The average age of the women was 46.6 years compared with 55.8 years for the men. Of the twenty-four patients in whom pulmonary emboli developed, thirteen were women and eleven were men. Of the thirteen women in the "embolic" group, seven

TABLE I
ANTECEDENT SHOCK AND HYPOTENSION IN POSTOPERATIVE THROMBOPHLEBITIS

Group	No. of Patients	With Shock or Hypotension	With Shock Alone
Postoperative thrombophlebitis	98 (51% were 50 yr. or older)	52*	31†
Control series of major surgical procedures	2,000 (54% were 50 yr. or older)	439*	267†

*$x^2 = 31$, $N = 1$, $P < 0.0001$. The difference between the two groups is significant if P is 0.05 or less.
†$x^2 = 25.6$, $N = 1$, $P < 0.0001$. The difference between the two groups is significant if P is 0.05 or less.

(53 per cent) were below fifty years of age, and six (47 per cent) were fifty years or older. In striking contrast to this is the male "embolic" group of eleven patients of whom one (9 per cent) was below fifty years of age, and ten (91 per cent) were fifty years or older. Four of the patients, two men and two women, died from pulmonary embolism. Their ages ranged from sixty-two to seventy-one years. This constitutes an incidence of fatal pulmonary embolism of 16.6 per cent in the "embolic" group and 4 per cent in the entire group of ninety-eight patients.

The type of anesthesia used was not related to the occurrence of shock or hypotension. In the "no shock" group thirty spinal anesthesias were given compared with thirty-two spinal anesthesias in the "shock" group. Nitrous oxide, cyclopropane and ether were administered singly or in combination to fourteen of the "no shock" group and to thirteen of the "shock" group. Miscellaneous types of anesthesia were used in the remainder of the two groups.

In fifty-two (53 per cent) of the ninety-eight patients shock or hypotension had occurred, and in forty-six (47 per cent) neither shock nor hypotension was noted. There was a significantly higher incidence of shock and hypotension in the group with postoperative thrombophlebitis when compared with a control group of 2,000 major operations on patients of approximately the same age as those affected by postoperative thrombophlebitis. (Table I.) The incidence of shock alone was much higher in the "thrombophlebitis" group than in the control group. (Table I.)

The operations which were followed by thrombophlebitis are shown in Table II. The incidence of shock and hypotension was high in most instances; thus, of six partial gastrectomies for peptic ulcer, shock or hypotension occurred in four. Of eleven hysterectomies performed for benign disease, six were associated with shock or hypotension. All three splenectomies in this series were accompanied by shock or hypotension. Ten appendectomies were performed for acute appendicitis with or without rupture of the appendix, and in four of these shock or hypotension occurred during operation. What is the expected incidence of shock and hypotension in appendectomy performed for acute appendicitis? In a randomly selected series of 766 appendectomies performed for acute appendicitis, fifty-two (6.7 per cent) were accompanied by operative shock or hypotension. This may be contrasted with a 40 per cent incidence of operative shock or hypotension in the ten appendectomies of this series which were followed by thrombophlebitis.

The time of onset of clinical evidences of thrombophlebitis varied from one day to thirty-eight days after operation, the average time for the entire group being ten days. When the group of ninety-eight patients was divided into those with antecedent shock, antecedent hypotension and no antecedent shock or hypotension, the times of onset of thrombophlebitis did not differ significantly between the three subdivisions. The presence of antecedent shock or hypotension did not seem to accelerate the time of onset of clinical thrombophlebitis. The duration of shock and hypotension varied from ten minutes to twenty hours. There was no significant relationship between the duration of shock and the likelihood of pulmonary embolism.

COMMENTS

As in other reported series of postoperative thrombophlebitis, there is a preponderance of women in the present group of patients. Appar-

TABLE II

OPERATIONS FOLLOWED BY THROMBOPHLEBITIS IN THE LOWER EXTREMITY

Operation	Shock or Hypotension	No Shock	Operation	Shock or Hypotension	No Shock
Hysterectomy (fibroids, pelvic inflammatory disease)	6	5	Diaphragmatic hernioplasty	1	1
Cesarean section	2	3	Partial gastrectomy (carcinoma)	1	2
Ectopic pregnancy	2	1	Closure of perforated peptic ulcer	3	2
Abdominoperineal resection (carcinoma)	2	0	Appendectomy (acute appendicitis)	4	6
Ileocolostomy	0	1	Lysis of adhesions	1	1
Resection of rectum (stricture)	1	0	Exploratory laparotomy for inoperable carcinoma (stomach)	1	0
Nephropexy	0	1	Witzel ileostomy	0	1
Cystostomy	0	1	Ligation of gastric and splenic arteries (cirrhosis)	1	0
Resection of bladder	1	1	Gastroenterostomy (ulcer)	0	1
Prostatectomy	1	4	Pyloroplasty	0	1
Thoracotomy (gunshot wound)	2	0	Repair of intestines (gunshot wound)	2	1
Thoracolumbar sympathectomy	1	0	Cholecystectomy	2	1
Nailing of hip	0	1	Common duct exploration	1	1
Radical mastectomy	1	1	Cholecystojejunostomy	1	0
Dehiscence repair	1	1	Colon resection	2	0
Hernioplasty	1	4	Colon resection (carcinoma)	1	1
Partial gastrectomy (ulcer)	4	2	Colostomy (carcinoma)	3	2
Splenectomy	3	0			

ently this is not due to a sex-linked factor but rather to the fact that women are more frequently subjected to operations which are followed by a relatively greater number of instances of thrombophlebitis. If the operations on the sex organs of each sex are excluded (prostatectomy in men and pelvic and breast operations in women), the number affected by thrombophlebitis after operations on "nonsexual" organs and tissues is the same in men and women. (Table III.)

The data presented herein indicate that antecedent shock and hypotension occur in a statistically significant number of patients in whom thrombophlebitis develops in the lower extremities following surgery. That shock may result in thrombus formation in other parts of the vascular system, for example the coronary arteries, seems to be well established.[3] Because of its high incidence in patients with postoperative thrombophlebitis, it is suggested that antecedent shock may be a factor in the causation of this condition. If this is true, the nature of the etiologic action of shock is completely unknown. Possible underlying mechanisms have been discussed elsewhere.[2]

The multiplicity of the factors entering into the pathogenesis of thromboembolism has been stressed in a recent review.[1] Some of these factors are known; others are not. Sometimes they appear to exert a thrombosis-producing effect, and at other times they do not. The results of the present investigation indicate that an additional mechanism may be present, the activity of which determines whether or not various etiologic factors will be effective in producing venous thrombosis. This mechanism may be the capacity of the affected person to re-establish and maintain circulatory homeo-

TABLE III

RELATION OF SEX TO POSTOPERATIVE THROMBOPHLEBITIS

Group	All Operations	Operations on Sex Organs	Remainder	With Pulmonary Embolism
Men	40	5 (prostatectomies)	35	9
Women	58	21 (pelvic operations, radical mastectomy)	37	10

stasis following a period of general or local disturbance of the circulation.

SUMMARY AND CONCLUSIONS

A series of ninety-eight patients with postoperative thrombophlebitis has been studied. A control group of 2,000 patients who had been subjected to surgery was used for comparison. The incidence of shock and hypotension before, during and after operation was significantly higher in the patients with postoperative thrombophlebitis than in the control series of 2,000 surgical patients. The possibility that shock may be a factor in the etiology of thrombophlebitis is discussed.

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The Critical Point of Cooper's Ligament Hernia Repair

ALTERNATIVE TECHNICS FOR ITS REINFORCEMENT

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THE problem of anatomic weaknesses in the inguinal region is a perpetual one. Despite innumerable technical advances in hernia repair, the goal of perfection has not been attained. A variable but substantial incidence of recurrence is still reported from our largest surgical clinics. Those surgeons particularly interested in the problem of hernia repair are not content to accept unequivocally all the presently recognized standard operative procedures as the ultimate in hernia progress. For instance, there is cumulative clinical and statistical evidence to affirm the existence of a potentially inherent weakness in one of the most reliable and effective operative procedures, Cooper's ligament hernia repair. The attainment of greater reparative success is dependent upon alertness of the surgeon in recognizing any potentially vulnerable area, and also upon his resourcefulness in devising methods essential for correction.

It is generally accepted that the most consistently weak area in the classic operation of herniotomy (the Ferguson or Halsted II operation) is either at the base of the intermediate inguinal (Hesselbach's) triangle or at the pubic angle of the posterior wall of the canal. This is attributed to: (1) the narrowness of the rectus muscle, which widens the inguinal region, at the pubic angle; (2) the increased intra-abdominal pressure which focalizes medial to the inferior epigastric vessels;[10] (3) the direct insertion of the obliquus abdominis internus and transversus abdominis onto the rectus sheath without an intermediary aponeurotic stratum and at a variable distance from the pubic spine; (4) a rudimentary conjoined aponeurosis often associated with an asthenic habitus; and (5) deficient density of the fascia transversalis. In those patients having rudimentary conjoined aponeuroses the medial posterior wall of the canal is deprived of the normal conjoined tendon support. In such instances the medial triangular area is for the most part unilaminar. Consequently, the security of the posterior wall of the inferior angle of the inguinal region in such aplasia is wholly dependent upon the integrity of the fascia transversalis.

Inguinal ligament hernia repair with funicular transposition (herniorrhaphy) is predicated on the strengthening of the medial floor by aponeurotic imbrication with resultant lesser potentiality for recurrence in this area, yet this technic simultaneously creates greater susceptibility to weakness at the abdominal ring by the creation of a joint portal of exit for the cord. Because of the almost universal high regard for over half a century for the Bassini and Halsted I technics the surgeon may be inclined to be less wary of this type of inguinal ligament repair and be lulled into a false sense of security regarding prognosis of the reinforced wall.

Cooper's ligament hernia repair possesses many advantages over the conventional inguinal ligament herniorrhaphy. In the former the parietal wall is more deeply placed, actually becoming intrapelvic, with resultant better diversion and wider dispersion of the intra-abdominal force. Because of the more favorable shunting angle produced by the altered relationship of the abdominal wall there is less concentration of the intraperitoneal pressure on the primary suture line. Coincidentally, the interligamentous area and the trihedral space are excluded. With the addition of the recently introduced combined maneuver, a wide vertical synthesis of the parietal and the turned down ligamentous walls is attained, creating in effect a central tendon with increased solidity.

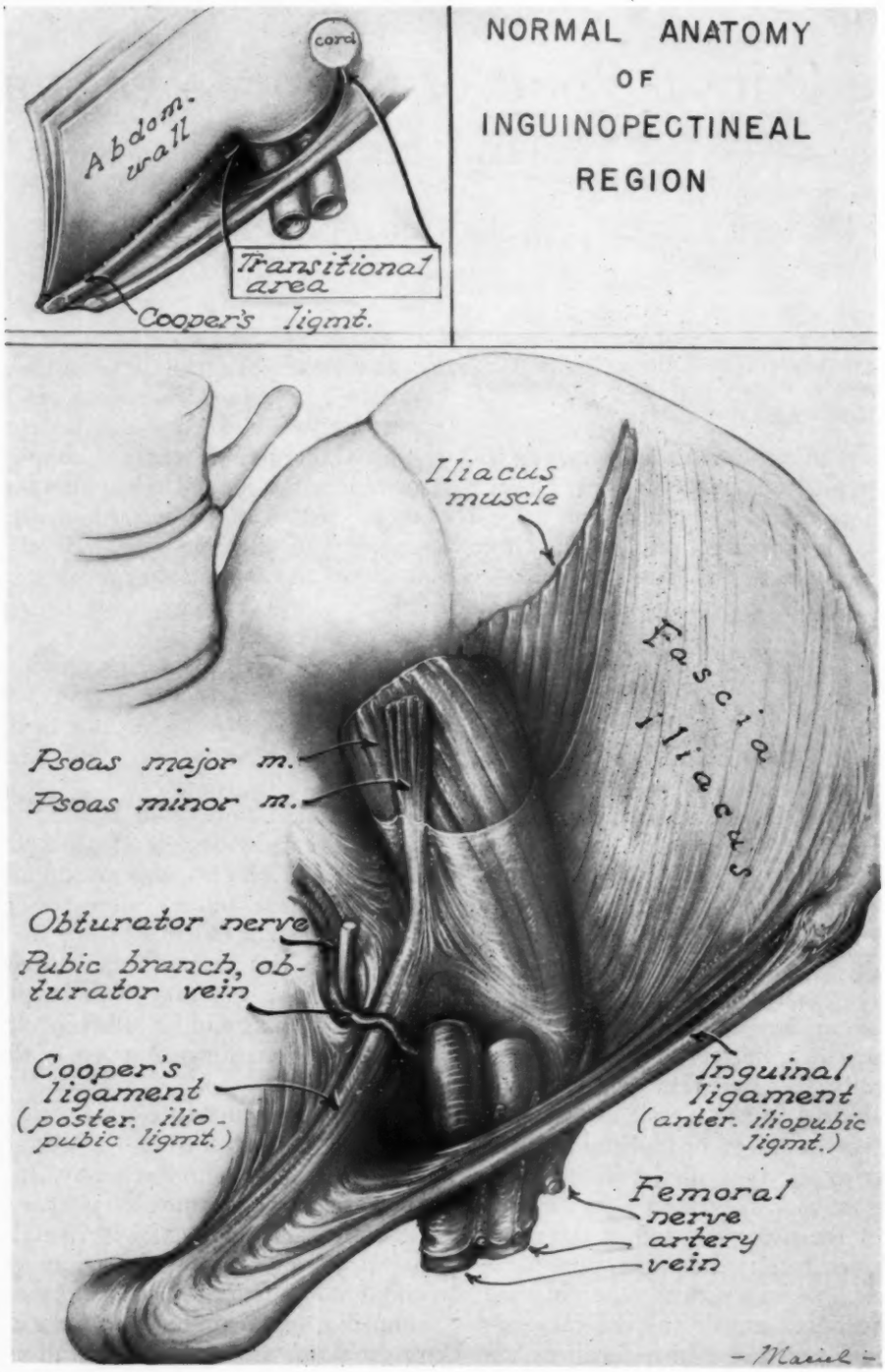

FIG. 1. Relationship of anatomic structures of inguinopectineal region and adjacent pelvic fossa. The pubic branch of the obturator vein which communicates with the external iliac vein is commonly present and invariably requires ligation for complete exposure of Cooper's (posterior iliopubic) ligament. Insert depicts transitional area. Its juncture with Cooper's ligament is important strategically and is often referred to as the transitional angle.

There is also dual insertion of the parietal wall and of the inguinal ligament into the internal and anterior surfaces, respectively, of Cooper's ligament which serves as a means of further cushioning for the intra-abdominal pressure.[2] Since Cooper's ligament triple maneuver repair possesses so many attributes not inherent in inguinal ligament repairs, it is difficult to understand how this widely accepted procedure could have a potential weakness.

From careful follow-up studies of more than 900 patients upon whom Cooper's ligament repair[3] has been performed, it has been shown that the critical area is not in the reinforced floor of the canal or at the abdominal ring but is located at the junction of Cooper's ligament component of the repair with the transitional component which is vertical to it. The precise pivotal area has been designated the transitional angle. It is therefore essential that the interligamentous space at this junctional angle be recognized and implemented by the strongest possible laminar anchorage at this point. (Fig. 1.)

The transitional area not only encompasses the critical angle but also comprises a variable-sized occult space extending from Cooper's ligament subjacent to the external iliac vein anteriorly and continuing superiorly to the abdominal ring. Actually this potential space is invariably present but its more important deeper interligamentous variant is not exposed and consequently is unrecognized in the typical inguinal ligament repair. In contrast, with the complete exposure of Cooper's ligament preparatory to its utilization for parietal anchorage, the space medial and suprajacent to the external iliac vessels becomes quite obvious. This space is also bounded superiorly by the arcuate portion of the obliquus abdominis internus and inferiorly by the lowermost fibers of its cremaster division and by the velamentous pectineal fascia bridging the interligamentous space.[5]

With increasing experience gained from Cooper's ligament hernia repair, we are gradually abandoning the former single maneuver unilaminar repair of Lotheissen[11] and the subsequent popular bilaminar technic of McVay and Anson[13] in favor of the triple maneuver procedure which we prefer to designate as the combined operation.[3] The reason for the adoption of the latter procedure stems from a statistical study of the anatomic sites of the recurrences. In one follow-up study of 117 interligamentous hernias which were repaired by the standard single maneuver technic there were twelve recurrent hernias, ten of which were at the transitional area.[4] This revelation was sufficiently convincing so that in order to attain a further reduction in the incidence of recurrence by this technic it was decided that more attention must be focused on devising means of reinforcing the angular component of the transitional space. There can be small doubt that the strengthening of this area is the most crucial step strategically in the success of Cooper's ligament repair, as evidenced by a successive reduction of recurrent hernias in a later series to 3 per cent. Consequently, there seems ample justification for a discussion of the various evolutionary procedures which have been proposed for augmenting this vulnerable area at the transitional angle. Moreover, it is reasonable to assume that if the criteria regarding the applicability of these technical modifications were more widely known and easily available in the literature, certain surgical pitfalls could be avoided and a more solid repair confidently anticipated.

Based upon the experience in our clinic the most common technical error is the failure to obtain adequate security of the suture at the transitional angle in Cooper's ligament hernia repair. This fact cannot be too strongly emphasized. The prerequisites which must be fulfilled in parietoligamentous anchorage at the transitional angle are: (1) use of the internal or vertical portion of Cooper's ligament; (2) the attainment of widest possible appositional contact; (3) augmenting the security of the suture by using adjacent unyielding structures; and (4) the molding of the wall around the medial aspect of the external iliac vein to obliterate any residual subvascular triangular space which may subsequently serve as a nidus for recurrent hernia.

Ordinarily a good hold can be obtained by passing the needle through the usually dense vertical portion of the ligament. (Fig. 2.) However, the ligament may occasionally be so attenuated as to predispose to inadequate anchorage of the suture. When this occurs, the needle may tear the sparse ligamentous fibers from their attachment to the internal surface of the superior pubic ramus. Another equally potent cause of insecurity is the placement of a series of sutures in the same horizontal

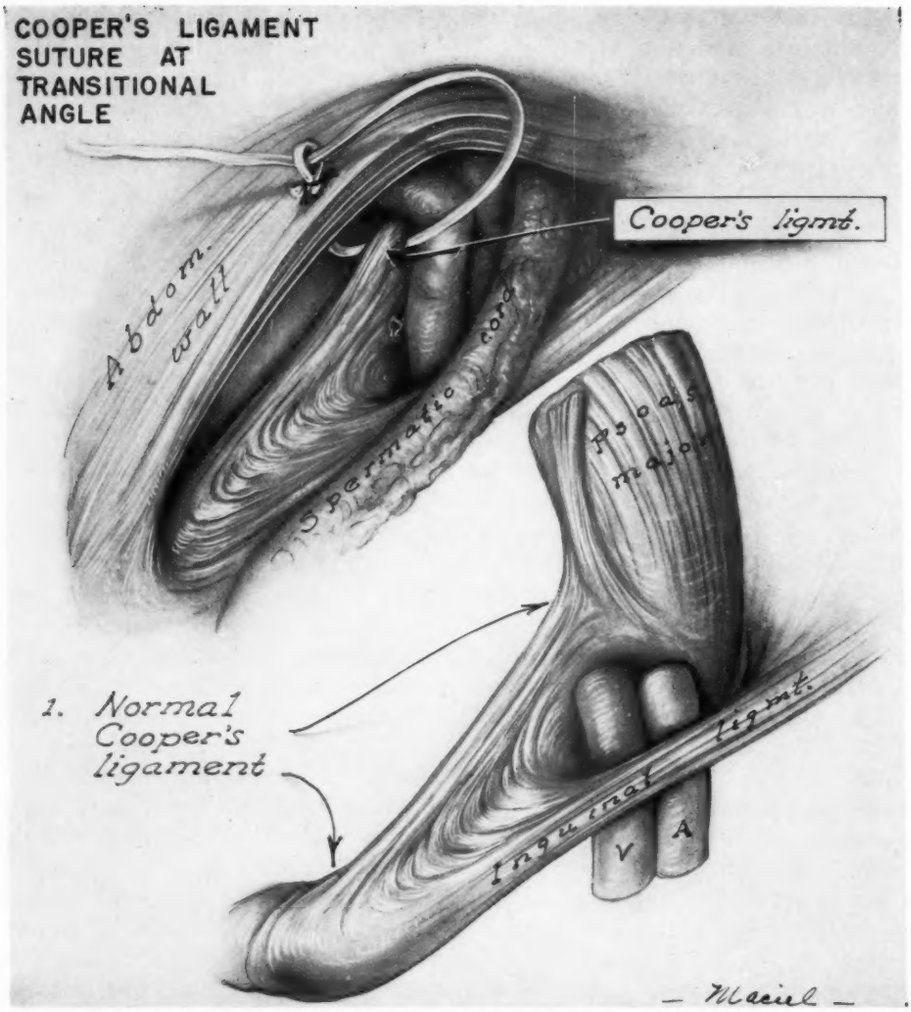

FIG. 2. Illustrates introduction of the first suture in Cooper's ligament hernia repair. This passes through the triple parietal strata and through Cooper's ligament subjacent to the external iliac vein. This suture closes the widest space in the posterior wall of the inguinal canal (trihedral space); upon this the maximal stress of the repair is imposed. The loop method of transfixion of the fascial suture as shown is considered preferable.

plane of the ligament instead of staggering their level of emergence. Such injudicious practice may have the effect of splitting a normal Cooper's ligament at the juncture of its vertical and anterior components. Confronted with either of these eventualities, the resourcefulness of the surgeon may be taxed to its fullest in improvising a method for fixation of the parietal wall at this crucial point.

In those rare instances in which the vertical portion of Cooper's ligament becomes loosened or detached by the placement of multiple sutures in the same horizontal plane, it may be necessary although less desirable to use the anterior component of the ligament in lieu of the vertical portion. The former provides edge contact in contrast to the more desirable wide surface contact which obtains when the wall is apposed to the vertical surface of the ligament.

ALTERNATIVE METHODS IN LIEU OF DEFICIENT COOPER'S LIGAMENT

In reviewing the literature three technical procedures have been found which have been proposed as substitutes for Cooper's ligament deficiency. They are: (1) psoas minor tendon; (2) psoas fascia; and (3) obturator foramen (circumpubic). I wish to propose another, the lateral transpubic suture.

Psoas Minor Tendon Method (*Fig.* 3). A comparatively new and quite practical alternative method is the insertion of the pivotal

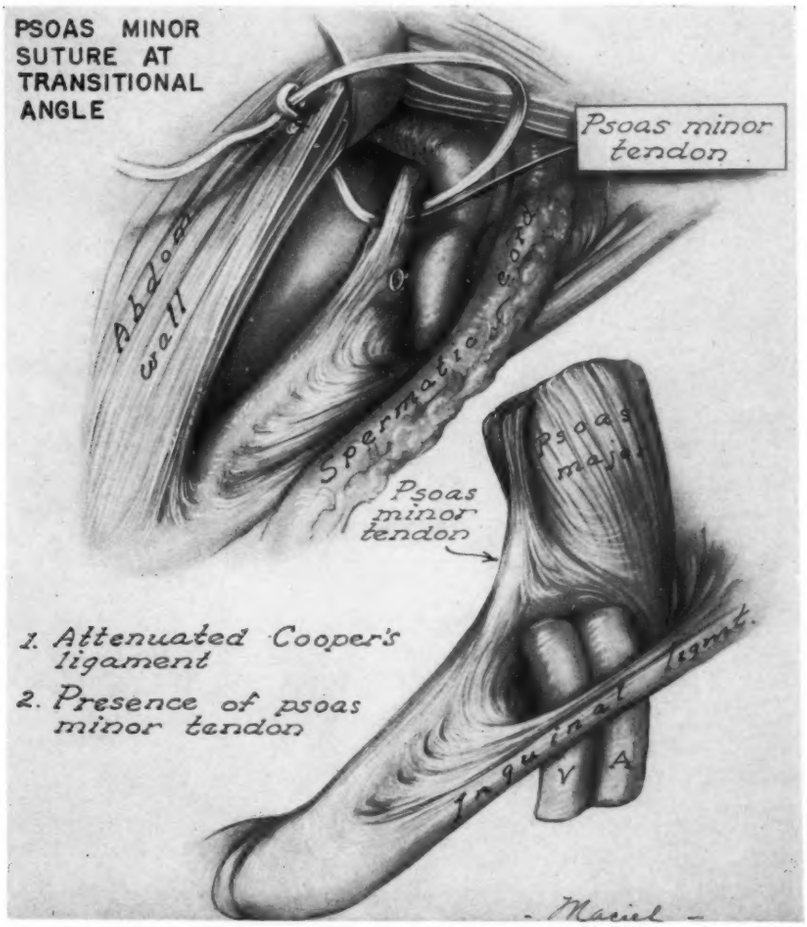

FIG. 3. In the absence of a sufficiently dense Cooper's ligament for secure anchorage of the suture the tendon of the psoas minor makes an ideal substitute. Unfortunately, agenesis of the psoas minor is not uncommon. Occasionally it may be possible to utilize the ligament and tendon jointly.

transitional suture through the psoas minor tendon at its tendopectineal juncture. This maneuver is ideally suited for lateral ligamentous attenuation and should invariably be the surgeon's first choice provided the minor tendon is present. The suture when so placed lies slightly posterior to the medial edge of the external iliac vein. When the suture is drawn taut, it obliterates the potentially vulnerable interligamentous infundibuliform space by apposing the parietal wall to the psoas minor tendon and often obtains some contact with the subjacent Cooper's ligament. If this maneuver is done properly, the parietal wall should follow the contour of the external iliac vein but without displacing or compressing it. Occasionally it may be feasible to place the suture in a thin Cooper's ligament and consecutively pass the needle through the psoas minor tendon which in effect makes a dual insertion of the suture and also gives a broader contacting surface for the approximated wall. The combined tendoligamentous suture is recommended in all Cooper's ligament repairs in which the psoas minor tendon is present.

A preparatory prerequisite to facilitate performance of this simple technical variant is the total exposure of Cooper's (posterior iliopubic) ligament and the identification of the psoas minor tendon. (Fig. 1.) The most frequently encountered obstacle to the use of this tendon stems from the fact the psoas minor muscle is often congenitally absent. However, it has been our experience that when the tendon is diligently sought for, it will be found more often (in 10 per cent of our last 100 repairs). I am convinced that before adopting the technic of total exposure of the internal vertical portion

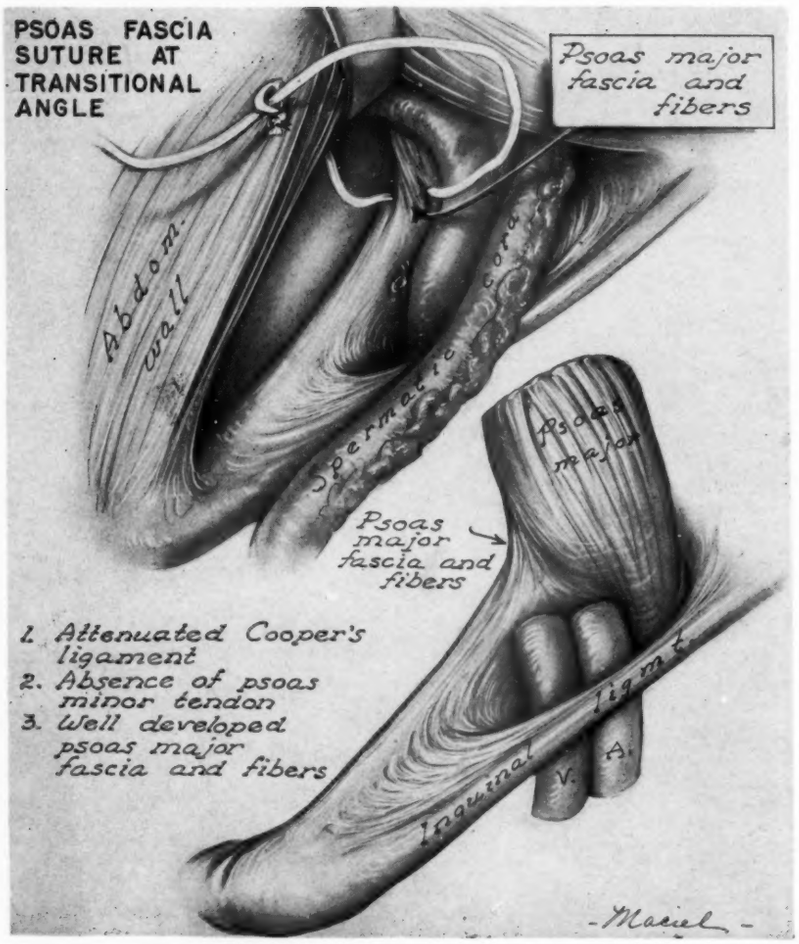

FIG. 4. When confronted with agenesis of Cooper's ligament and of the psoas minor muscle the fascia covering the psoas major muscle offers the next most feasible site for fixation of the suture. Obviously, a suture through the psoas fascia and fibers will be more anteriorly placed than in the two previously illustrated technics of using either Cooper's ligament or psoas minor tendon.

of Cooper's ligament many psoas minor tendons were missed because of inadequate exposure of the former occult funnel-shaped area, presumably on the false premise of injuring the obturator nerve or of causing vascular trauma. To facilitate exposure of the psoas minor tendon, Rees[16] advocates abduction of the hip which also affords protection to the external iliac vein by causing its lateral displacement. However, we have not found it necessary to resort to this maneuver in order to obtain adequate ligamentous exposure.

Psoas Fascia Maneuver (*Fig.* 4). Another method of securing the parietal wall at the transitional angle is to pass the needle carrying a suture through a sizable bundle of fascia and fibers of the medial portion of the psoas major muscle as it crosses somewhat obliquely the posterior portion of the superior pubic ramus. We have not observed agenesia of the psoas major muscle. The psoas fascia in some patients is dense, creating confidence in the resistance and security of the suture; in others the fascia is thin, and often the underlying fascicular fibers are friable, engendering doubt as to whether the wall so anchored will withstand inordinate intra-abdominal pressure. Therefore, the lack of uniform density of the psoas fascia, the fact the wall is not as deeply placed and the absence of ligamentous apposition are factors which militate against the employment of this variant. The conjoined insertion of the suture into a remnant of Cooper's ligament and the psoas fascia is not possible because the points of anchorage are too widely separated. This method in our opinion is rarely feasible.

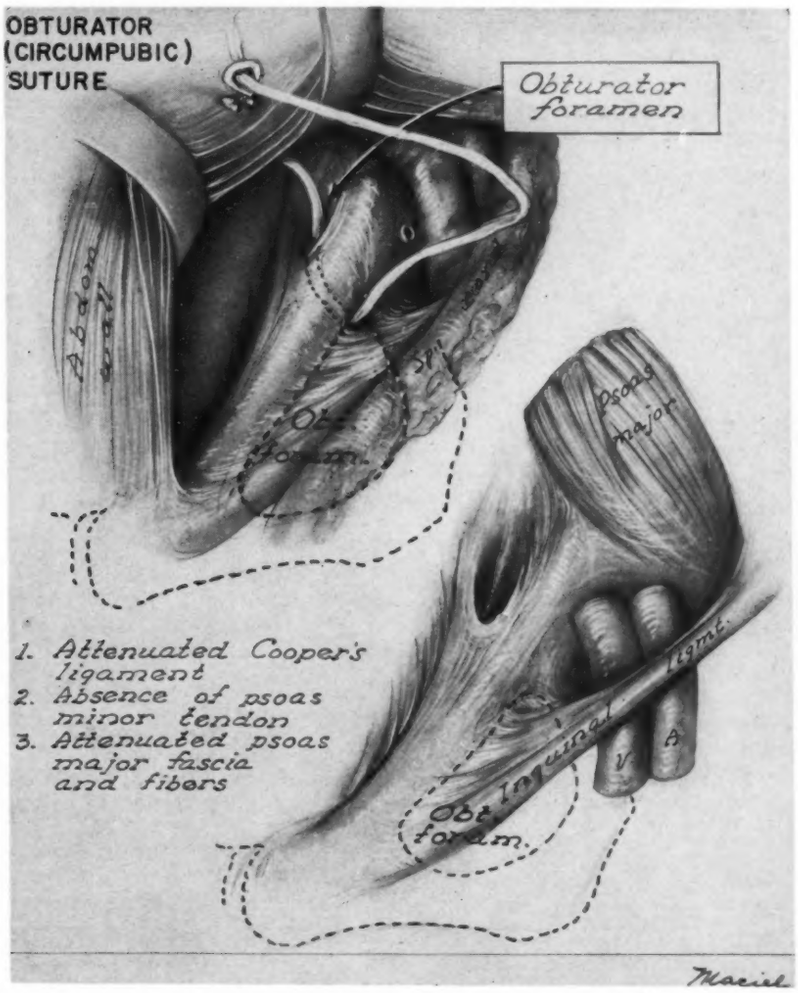

FIG. 5. The use of the obturator foramen through which to pass a suture, staying close to the superior pubic ramus, is illustrated. The difficulty encountered in the placement of this suture and the possibility of puncturing the obturator nerve are the chief objections to the use of this technic.

Obturator Foramen or Circumpubic Suture Procedure (*Fig. 5*). A unique and ingenious idea of stabilizing the parietal wall laterally was conceived by Cavazzani.[6] He proposed passing a suture through the obturator foramen at its most lateral boundary, hugging and encircling the superior pubic ramus. It is important to visualize the structures of the obturator canal so as to avoid injuring them. At first it would seem that this technical variant may have some merit, but on more careful consideration several objections to the employment of this maneuver are evident. The obturator foramen is considerably medial to the transitional angle so that the combined wall is drawn deeply enough but away from the focal point of the subvascular weakness, in effect, increasing the size of the vulnerable area without reinforcing it. Despite precise anatomic knowledge and meticulous care required in placing this suture there is a remote possibility of injury to the obturator nerve or puncture of the obturator vessels. In our experience we have not had occasion to use this variant, and for the reasons cited I would be very reluctant to resort to this means of implementing the critical area. Moreover, I have not been able to find any reports in the literature stating that this technic has been employed by other surgeons.

Lateral Transpubic Suture Method (*Fig. 6*). It is axiomatic that when several methods are available for the treatment of a surgical entity none is wholly satisfactory. We agree with

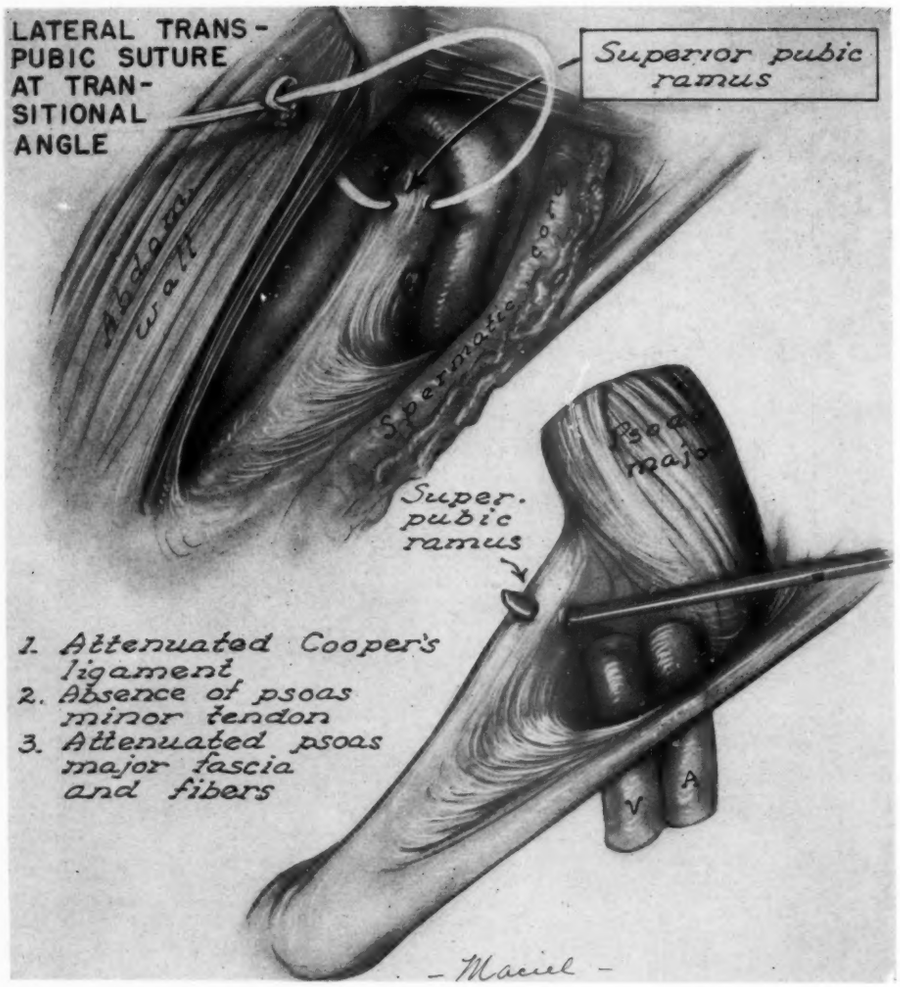

FIG. 6. In the absence of adequate ligamentous, tendinous or fascial structures in which to anchor the pivotal suture, a transosseous canal through the superior pubic ramus at this site for the passage of the suture should be considered. An ordinary trocar is inserted so that it just penetrates the anterior cortex, through which an angular pointed instrument is made to perforate the internal surface of the ramus, as shown. The fascial suture is then threaded through the bony canal.

Halsted[9] and Fallis[7] that every hernia merits the very greatest care and the strongest possible closure at the time of the initial repair. Our objective is to present a new type of suture anchorage of the parietal strata to overcome the potential anatomic weaknesses which may exist at the transitional angle. The anatomic deficits which may jeopardize the success of the operation are: (1) lateral deprivation of the vertical and/or anterior components of Cooper's ligament; (2) concomitant absence of the psoas minor tendon; and (3) attenuated psoas major fascia. In order to cope with these infrequent but vitally important structural deficiencies we devised a new technic of employing a transpubic suture which passes through the lateral pubic shelf underlying the thin anterior and vertical components of Cooper's ligament. The internal bony ledge follows the course of the iliopectineal line and is easily identified. The excellent results obtained in the cases in which this suture has been used have prompted this preliminary report which includes a description of the technic.

TECHNIC

An important prerequisite of the transpubic suture technic is the complete exposure of Cooper's ligament from the pubic spine to the iliopectineal eminence. The entire ligament should be visualized and palpated. If this is

practiced regularly in all Cooper's ligament repairs, the surgeon will learn to detect variations in the fascial structures overlying the iliopectineal shelf of the superior pubic ramus. He will learn to interpret the normal density of Cooper's ligament better and will be less likely to overlook the psoas minor tendon which may not stand out separately but may infrequently be incorporated in the psoas major muscle. These observations, if made routinely and thoroughly, may result in discovering structures adequate for parietal anchorage, which would preclude the use of more elaborate technical deviations.

The most important and constant vessels encountered in the exposure of the posterior iliopubic (Cooper's) ligament are the inferior epigastric artery and vein. If the hernia has produced appreciable dilatation of the abdominal ring, the vessels may be displaced medially. They are freed for the width of the posterior wall of the canal, a distance of perhaps 2.5 cm. This vascular segment is excised and the ends ligated with transfixion sutures. The purpose in removing such a long segment is to preclude puncturing the vessels proximal to the stump when a deep and generous stitch is taken through the trilaminar wall in the first maneuver of the repair.

Continuing the process of total ligamentous exposure, the attenuated fascia transversalis is incised along the junction of the posterioinferior boundary of the trihedral space or canal including its variants, the posterior medial (Henle's) ligament and the anterior medial ligament or ligamentum inguinale reflexum; proceeding lateralward, any remnants of the interfoveolar ligament of the floor of the canal are divided. Bisgard[1] believes that the anterior reflex ligament is constant and of such uniform density that the pubic angle which it spans is secure; he thus advises against incising it. This may be true, but division of the reflex ligament gives better exposure of the inferior or pectineal angle and definitely facilitates the suturing of the parietal wall to the entire extent of Cooper's ligament. We have frequently found two or three small veins crossing the medial half of the superior pubic ramus. These should be ligated. Only occasionally does a small artery accompany the vein. We have been struck by the frequency of a rather large vein about the size of the inferior epigastric, crossing the superior pubic ramus laterally and emptying into the external iliac. It has occurred in perhaps 10 per cent of the cases in our series. When present it must be ligated to obtain lateral exposure of the ligament. The obturator artery may arise from the hypogastric, the external iliac or the inferior epigastric. Its anomalous position has been observed only a few times in the Cooper's ligament repairs done at this clinic. In each instance the artery crossed the pubic ramus at the junction of its posterior and middle thirds. When this anomaly is present, ligation of the artery is necessary to gain adequate lateral ligamentous exposure. The obturator nerve enters the pelvis obliquely and follows the inferior edge of the superior pubic ramus until it enters the obturator canal. It is almost invariably visualized in the exposure of the posterior third of Cooper's ligament. It has not been injured in our rather wide experience with Cooper's ligament repairs. In exposing the posterior third of the ligament, the insertion of the psoas minor tendon can be visualized but is more certainly identified by palpation if it is present. It lies in the horizontal plane parallel and deep to the major muscle. The importance of the psoas minor tendon as alternate anchorage of the most lateral suture of the first maneuver has already been stressed. Another point worthy of mention is the occasional presence of a sizable chain of lymph nodes which lie inferior and adjacent to the external iliac vein. The removal of these nodes if appreciably enlarged will eliminate the possibility of extraneous tissue being interposed between the parietal wall and Cooper's ligament.

Normally Cooper's ligament is a dense, fixed, constant, fibrous membrane having two components: (1) the vertical, which is that portion intimately intermeshed with the periosteum of the internal vertical aspect of the superior pubic ramus and (2) that portion which overlies the more accessible anterior aspect of the ramus. We are indebted to Seelig and Tuholske[17] for a summary of the many divergent views of anatomists regarding the role of the various fascias in the formation of Cooper's ligament. Suffice it to say this lack of unanimity still prevails. However, these fasciae minutae are not particularly important to the surgeon.

The anterior surface of the superior pubic ramus is bared for about 1 cm. at the medial

edge of the psoas major muscle. With an ordinary trocar a small hole is made through only the anterior cortex of the bone. This can be done with moderate pressure on the instrument. The counter opening on the internal surface of the ramus must be at almost a right angle to the first hole. We first tried using an angular dental drill in an effort to negotiate the proper angle; however, this was without success. After trial and error the end of a screw driver, about 1 cm. from its tip, was fashioned into a rounded point and bent to an angle of 90 degrees. The point of the curved instrument is placed in the anterior cortical opening and gently guided so that it rests against the middle of the internal cortical surface of the ramus. With steady lever-like action the cortex is easily perforated. (Fig. 6.) The instrument should be under complete control so as to preclude the possibility of fracturing the bony shelf between the two holes. Although such a complication is conceivable, it has not occurred in our experience. An acutely curved needle holder also may serve this purpose and has been used to make the internal hole. Should there be some oozing from the cancellous bone, a small pledget of gelfoam® is inserted into the osseous canal.

Because of its elasticity, greater tensile strength, ease of procurement and its viability we prefer an autogenous fascia lata suture. Although it has been repeatedly pointed out that the suture at the transitional angle is the most important strategically, one should in no way neglect any step designed to achieve a uniformly taut wall. The standard Cooper's ligament repair in this clinic is performed with two fascial strips taken from the ipsolateral thigh using a 2.5 cm. transverse skin incision through which the Masson stripper is inserted.[12] These fascial strips are autotransfixed on Gallie needles by either the Robins'[15] or Gallie and LeMesurier[8] technic depending upon the width of the strip. The distal ends of both sutures are ligated with silk, but the end of one is turned back on itself and tied so as to form a loop. It has been found that the suture slips more easily through the loop than when it is autotransfixed. Another advantage of the loop method of transfixion is that it further obviates the possibility of the suture splitting at its distal end under tension

The transpubic suture, which is the first to be placed, is the most lateral suture of the first maneuver of Cooper's ligament repair. It traverses the trilaminar parietal wall and then the previously prepared osseous canal. (Fig. 6.) When the suture is drawn taut, its facility and effectiveness in closing the infundibuliform space is quite striking. The second fascial suture is started superjacent to the pubic spine and continues through the parietal wall and Cooper's ligament, respectively, until reaching the first one. The two sutures are drawn taut, apposing the combined parietal wall to the vertical portion of the superior pubic ramus. Since the inguinal canal is a trihedral space, it is easier to bring the wall down at its apex, juxtalateral to the pubic spine and gradually progressing lateralward until reaching the transitional angle where the transosseous or first suture has been placed. The two sutures are interlooped at this juncture. The second one continues cephalad closing the transitional space; the other runs inferiorly and medially passing successively through the loops of the underlying previously placed fascial suture and through the inverted edge of the inguinal ligament which pectinealizes the latter. The converging V-shaped parietoligamentous planes are sutured together producing a vertically synthesized wall and in effect creating a central tendon, perhaps 1.5 cm. in height. This maneuver also serves as a protective perpendicular barricade at the transitional angle. The technic is identical with the standard triple maneuver Cooper's ligament hernia repair except the first suture traverses the lateral transpubic osseous canal.

Within the past year we have employed the lateral pubic transosseous suture technic in eight cases, with no complications. Follow-up examinations have consistently disclosed a uniformly unyielding resistant wall. It is fully realized that this is a very small series and that it will require five years or longer before sufficient data will be available for any valid conclusions on the definitive results of this technic. However, we believe that the lateral transpubic suture offers another means of gaining secure anchorage of the most important suture in Cooper's ligament technics.

COMMENTS

There are six potentially vulnerable areas of the inguinopectineal region. These areas are located at: (1) the inferior or pubic angle

which is frequently referred to as the internal inguinal triangle; (2) the base of the intermediate inguinal triangle which is perhaps better known as Hesselbach's triangle; (3) the abdominal ring or hiatus through which indirect hernias emerge; (4) the transitional angle which is the most frequent site of recurrence following Cooper's ligament repair; (5) the interligamentous area which is more frequently a concomitant rather than a solitary weakness; and (6) the parahiatal area lateral to the abdominal ring which may not evaginate through a definitive aperture but rather present as a dome-like weakness.

The first three areas enumerated ordinarily can be corrected by an inguinal ligament reparative procedure, while the latter three require a Cooper's ligament repair. The most strategic step in Cooper's ligament repair is the fortification of the wall at the transitional angle. The principle objective of this article is to focus attention on the potential weakness at the transitional angle in Cooper's ligament repair and to propose another method for attaining security and solidity of the parietal wall at this point. The alternative reparative variants, which have been described, have been so designed as to preclude the creation of a concomitant but unrecognized preclinical weakness.

Each respective evolutionary step which has been successively outlined for dealing with the anatomic fault at the transitional angle has been predicated on one or more coexisting, definitely demonstrable structural deficiencies. The technics devised present the most direct and rational approach consonant with recognized surgical principles. We should like to emphasize that when it is feasible, there is no objection to combining one or more of these alternative methods.

In searching the literature it was found that the transpubic suture is not wholly new. It was employed by Nicoll[14] in the radical cure of femoral hernia in 1903. However, Nicoll drilled the pubic ramus medial and inferior to the transitional angle. The suture was threaded through the drill hole and passed through the inferior edge of the inguinal ligament which when tied obliterated the femoral ring but without strengthening the susceptible transitional area. The operative procedure was performed through the inferior approach. In fact, the inguinal canal was not exposed, nor was Cooper's ligament visualized. Consequently, the technic which we have proposed differs from Nicoll's in principle, the definitive objective to be attained, location of the transosseous canal, the operative approach and the structures approximated.

CONCLUSIONS

1. The integrity of the transitional angle suture, which is the most lateral suture of the first maneuver in Cooper's ligament repair, is of crucial importance in preventing recurrence of the hernia.

2. The various alternative methods which have been devised for anchoring the transitional angle suture in lieu of a deficient Cooper's ligament have been described and the indications for each clearly set forth.

3. A new method using a lateral transosseous pubic suture offers another means of attaining security of the wall at the transitional angle, which incidentally is the most likely site of recurrence in Cooper's ligament technic.

4. Criteria for the employment of the proposed lateral transpubic technic have been outlined.

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We recommend:

Hernia. The Pathologic Anatomy of the More Common Hernias and Their Anatomic Repair. By Chester B. McVay, M.D., PH.D. A Monograph of Surgical Techniques Series. Edited by Alton Ochsner, M.D. 40 pages with 19 plates, each plate containing from 1 to 4 figures. Springfield, Ill., 1954. Charles C Thomas. *Price* $4.75.

Reproductive System. By Frank H. Netter, M.D. Volume 2, the Ciba Collections of Medical Illustrations, 286 pages. Edited by Ernest Oppenheimer, M.D. with a Foreword by John Rock, M.D. Commissioned and Published by Ciba Pharmaceutical Products, Inc., Summit, New Jersey, 1954. *Price* $13.00.

Clinical Biochemistry. By Abraham Cantarow and Max Trumper. Fifth edition, 738 pages, 54 figures, 99 page index. Philadelphia, 1955. W. B. Saunders Co. *Price* $9.00.

Purse-string Suture Repair of Indirect Inguinal Hernia

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DURING the past twenty years numerous improvements and refinements of surgical technic in the repair of inguinal hernias have been made. Most of these have been directed toward a more satisfactory repair of the floor of the inguinal canal medial to the inferior epigastric vessels (Hesselbach's triangle). The apparent result to most surgeons is a lowered incidence of "recurrent" direct hernias. During the period of popularity of the Bassini type of repair, in which the lower border of the internal oblique muscle was sutured to the edge of Poupart's ligament, it was common to note the appearance of a direct hernia some months or years following the repair of an indirect inguinal hernia. With the currently popular method of suturing the transversalis fascia either to Poupart's or Cooper's ligament, the formation of new or recurrent direct hernias has become less frequent. The recurrence of an indirect hernia, however, is still frequently seen, and the percentage of recurrence does not seem to have been lowered in recent years as much as the recurrence rate of direct hernias.

The purpose of this paper is to describe a method of repair of the internal ring which, in my experience, has markedly lowered the recurrence rate of indirect inguinal hernias.

In planning the repair of an inguinal hernia, one must be able to adjust his technical steps to the variations of anatomy and pathology encountered in the groins of different patients. However, certain basic principles hold true in every variation of indirect inguinal hernia: (1) The peritoneum alone is no barrier to the intra-abdominal pressure, being very easily stretched and poorly attached to the overlying transversalis fascia which allows it to slide and "pouch" readily through any defect in the adjacent fascia. (2) The transversalis fascia is the "first line of defense" and the most important barrier to the formation of a hernia. Its fibers form the edges of the internal inguinal ring. The edges of the transversalis fascia at the internal ring must be stretched apart in order for an indirect inguinal hernia to form. (3) The internal oblique muscle is an important "second line of defense," but only when it is physiologically capable of contracting strongly so as to stretch tightly across the inguinal canal floor in the internal ring area. (4) The external oblique fascia in its normal position in relation to the spermatic cord has little or no barrier effect against the formation of an inguinal hernia.

From the foregoing facts, the following principles in surgical treatment can be stated: (1) The ligation of the hernial sac at the internal ring is only important from the standpoint of presenting a smooth peritoneal lining to the intra-abdominal contents. (2) To prevent a recurrence of an indirect inguinal hernia the transversalis fascia must be approximated about the spermatic cord in a circular fashion which allows only enough room for the cord structures to emerge through it. If the transversalis fascial ring is unbroachable, an indirect hernia cannot recur. (3) The internal oblique muscle must not be damaged or displaced permanently from its normal position if its muscle fibers appear to be in a good functional state. If the muscle edge is atrophic or infiltrated with fat, it has lost its effectiveness as a barrier to the formation of a hernia. In such instances its substance should be replaced by a fascial flap, metal mesh or other suitable substitutes. (4) From the standpoint of the prevention of hernia recurrence, the external oblique fascia can be used to much more advantage by suturing its flaps across the floor of the inguinal canal rather than using it as a "roof" to cover the spermatic cord.

The most important principle in repairing an indirect inguinal hernia is to make certain the structures about the internal inguinal ring are sufficiently tight to prevent the first "tongue" of peritoneal sac from extending through the internal ring. This would seem to

be an unnecessary statement, but it has been my observation that many surgeons, when operating for indirect inguinal hernia, seem to lose sight of the fact that the hernia is at the internal ring and they concern themselves chiefly with reinforcing the tissues through

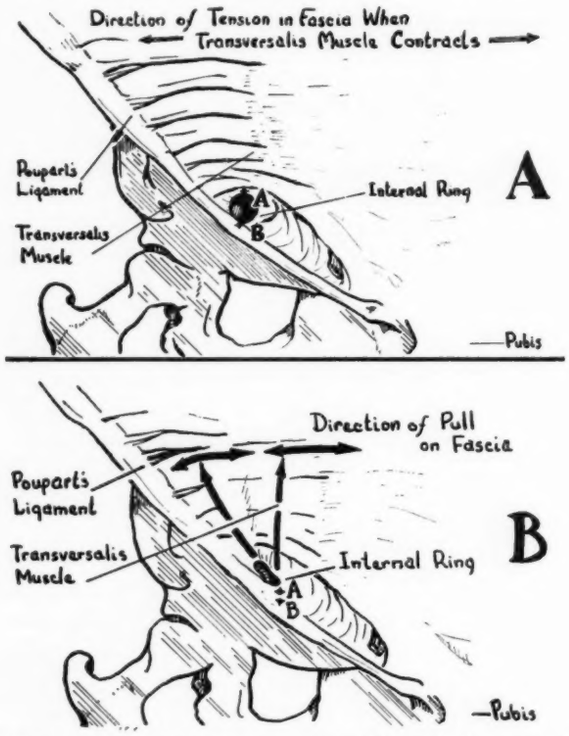

FIG. 1. A, illustrates normal positions (A) and (B) before sutures are placed for tightening internal ring in the common method of repair. B, after suturing points (A) and (B) together, tightening of the transversalis muscle during straining puts an oblique pull on point (A) away from point (B).

which a direct hernia occurs. The usual method for diminishing the size of the internal ring by suturing the transversalis fascia to itself or to the shelving edge of the inguinal ligament just medial to the exit of the spermatic cord from the internal inguinal ring has certain disadvantages. First, only a portion of the fascial borders of the internal ring is reinforced—the medial and superiomedial sides. If the cord can be shifted gradually lateralward and inferiorly away from the repaired area by repeated stress from increased intra-abdominal pressure, a recurrent hernia can occur between the repaired area and the shifted cord. Second, in placing sutures across the inguinal canal floor just medial to the internal ring, it can be noticed that the transversalis fascia is acutely angled across beneath the spermatic cord and there is usually some tension, as the most lateral suture is placed, to pull the medial transversalis fascia to the edge of Poupart's ligament. Later, when the patient coughs or strains, the constriction of the transversalis muscle causes tension and pull on the sutures anchoring this obliquely displaced fascia. The mechanics of this stress is illustrated diagrammatically in Figure 1. I feel certain that many recurrences are due to the most laterally placed sutures tearing through the tissue early in the postoperative period.

In order to prevent any shifting of the internal ring and to offer more complete support to the fascia about the exit of the spermatic cord from the internal inguinal ring, a heavy purse-string suture of silk is inserted through the transversalis fascia and the edge of the inguinal ligament, which completely encircles the spermatic cord. This may be done as a single-purse string suture, or two or more may be placed in concentric circles around the internal ring. The suture is tied tightly enough so that just enough room is left around the cord to allow circulation to and from the testicle. This suture not only pulls in firm fascial support around the internal inguinal ring but also gives a firm, non-distensible edge to the entire circumference of the internal ring.

TECHNIC

The indirect hernial sac is completely isolated from the spermatic cord and is closed at the internal ring in the usual manner. To accurately place the purse-string suture of heavy braided silk around the internal inguinal ring and spermatic cord, the entire circumference of the transversalis fascia about the cord must be exposed to view. This is accomplished on the medial and inferior border of the internal ring quite easily by simply elevating the spermatic cord from the floor of the inguinal canal. The superior portion can be exposed by retracting the edge of the internal oblique muscle. The lateral and superiolateral edges can best be exposed by clamping and cutting the cremaster muscle fibers as they cross from the internal oblique muscle onto the spermatic cord (Fig. 2) and then retracting the margin of the arching fibers of the internal oblique muscle laterally. When the entire fascial circumference about the cord at the internal ring is exposed and clear,

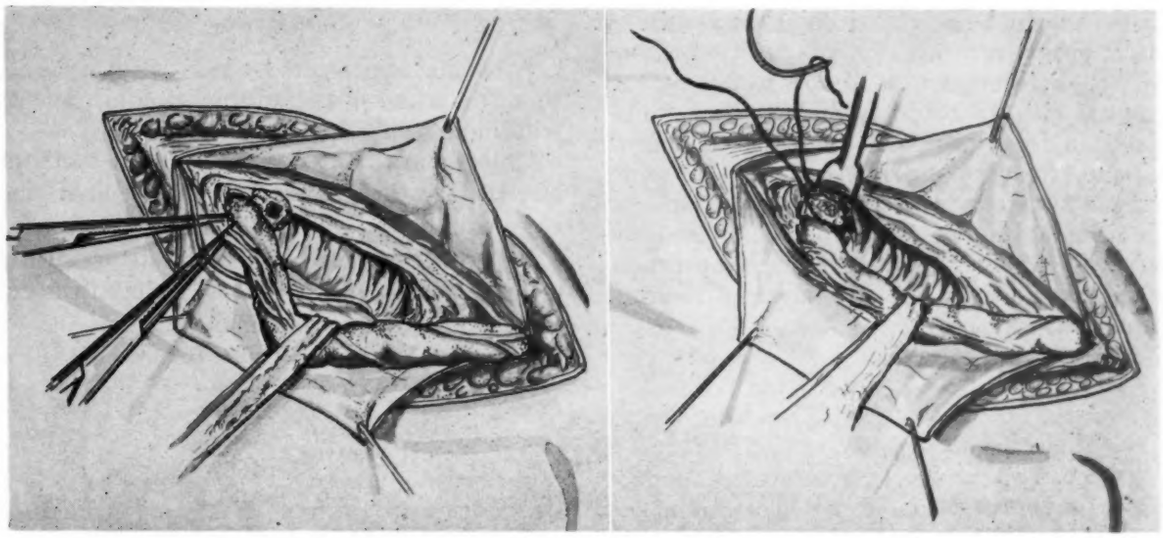

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FIG. 2. The cremaseter muscle fibers are clamped, cut and tied just lateral to the internal ring, where they are reflected from the internal oblique muscle onto the spermatic cord. The indirect hernia sac stump is demonstrated by upward retraction of the internal oblique muscle. The ileoinguinal nerve is preserved.

FIG. 3. The heavy purse-string suture of silk has been placed in the transversalis fascia about the neck of the hernia sac and exit of the spermatic cord from the internal inguinal ring, and is anchored to the shelving edge of Poupart's ligament below.

a No. 3 or No. 4 braided silk suture on a short round needle is started in the transversalis fascia superior to the neck of the previously ligated hernial sac and carried in and out of this fascia lateral to the spermatic cord, then through two portions of the shelving edge of the inguinal ligament, the second bite of the needle being about 3 mm. from the first one placed in the ligament, thence medially along the edge of the dilated internal ring back to the starting point. The suture must be accurately placed in the edge of the dilated internal ring, making a complete circle around the spermatic cord and neck of the hernial sac (Fig. 3) in order for the enclosed fascia to be pulled up snugly around the cord. The suture bites are placed about 5 mm. apart, and if they are accurately placed in the edges of the dilated internal ring, there will be no tension when it is snugly tied. I believe that tying the purse-string suture tightly enough is extremely important in this technic. The resident surgeons learning this technic are instructed to tighten the suture until they think it is too tight around the spermatic cord, and then tighten it just a little more. An opening through the purse-string suture, about 5 mm. in diameter, is sufficient to allow unimpaired circulation to and from the testicle.

RESULTS

All cases included in this series were operated upon before 1952. The known recurrences in a series repaired by the purse-string suture technic are compared with another series repaired by the Edmund Andrews principle. Of 274 cases in which the purse-string suture technic was used, there was one known recurrence of an indirect hernia. Of 248 cases in which the Edmund Andrews type of repair was used, there were eleven known recurrences. All of these recurrences were confirmed by reoperation as recurrent indirect hernias. The percentage of cases lost to follow-up study was essentially the same in the two series (29 and 33 per cent, respectively).

COMMENTS

Reoperation was necessary for the one recurrence in the series in which purse-string suture technic was used. The recurrent hernia developed through a purse-string opening 1 cm. in diameter. Obviously the suture had not been tied down tightly enough around the spermatic cord. It was interesting to note that the transversalis fascia around the edge of the internal ring and adjacent to the silk purse-string suture was exceptionally thick and strong—several

times as thick as the normal transversalis fascia in that area usually is. Apparently there is enough reaction around the suture at the internal ring to cause proliferation of fibroblasts adjacent to it with consequent thickening and strengthening of the fascia. This same thickening around the purse-string suture at the internal ring was also observed to be quite marked in another man undergoing reoperation for a direct hernia one year after an indirect hernia was repaired.

SUMMARY

A technic is presented which can contribute to a firm repair of the internal ring in patients with indirect inguinal hernia.

Objections to the commonly used methods of repairing the dilated internal inguinal ring are mentioned.

In my experience, the purse-string suture technic of repair has markedly reduced the recurrences of indirect inguinal hernia.

We recommend:

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Current Therapy, 1955. Latest Approved Methods of Treatment for the Practicing Physician. Edited by Howard F. Conn, M.D. Consulting Editors: M. Edward Davis, Vincent J. Derbes, Garfield G. Duncan, Hugh J. Jewett, William Kerr, Perrin H. Long, H. Houstan Merritt, Paul A. O'Leary, Walter L. Palmer, Hobart A. Reisman, Cyrus C. Sturgis, Robert H. Williams. (Active ingredients in commonly used products.) 692 pages. Philadelphia, 1955. W. B. Saunders Co.

The Problem of Duodenal Closure after Gastrectomy for Duodenal Ulcer

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PARTIAL gastrectomy is well established as a surgical procedure for the treatment of peptic ulcer. However, there are differences in opinion as to the advantages of various points in technic, such as the placement of the jejunal loop anterior or posterior to the colon, and the comparative value of the Hofmeister or Pólya types of anastomoses. The Billroth I procedure has been revived to some extent, but has limited value in the surgical treatment of the complicated duodenal ulcer.

This presentation will be limited to the technical problems involved in the closure of the duodenal stump after partial gastrectomy for duodenal ulcer, with particular reference to a method of duodenal closure developed by one of the authors (P. C.), and employed for the large posterior wall ulcer of the first part of the duodenum. When the duodenal ulcer is limited in size and relatively near the pyloric ring or in the second part of the duodenum, an adequate length of duodenum can be easily mobilized for the routine two- or three-layered closure. Frequently this is accomplished with an inner Connell suture of chromic catgut and an outer layer of interrupted Lembert or mattress sutures of No. oooo silk. A few additional sutures of silk are used to approximate the soft tissues over the pancreas to the duodenum. The blood supply to the duodenal stump must be adequately preserved, and the closure must be accomplished without undue tension.

If the ulcer is near the common bile duct, and it appears that the dissection or the duodenal closure may compromise that structure, the insertion of a T-tube into the common bile duct, as recommended by Marshall,[1] may be advisable.

The large ulcer involving the anterior wall of the first part of the duodenum presents a serious problem. The associated induration makes inversion of the duodenal wall very difficult. Closure may, however, frequently be accomplished if the duodenal stump is closed with interrupted Lembert or mattress sutures of silk and these sutures are placed only partly through the thickness of the duodenal wall.

The two-stage Finsterer type of resection has been employed when the ulcer was associated with considerable surrounding inflammatory reaction and induration. In recent years the use of a catheter in the duodenal stump has been employed as a temporary measure by Welch[2] and Priestley and Butler[3] and has proved relatively satisfactory.

The ulcer of the posterior wall of the first part of the duodenum frequently involves the duodenum to such an extent that after dissection an inadequate or unsatisfactory posterior duodenal wall remains for the closure. It may be considerably edematous, indurated or extremely short. Again, the dissection breaking into the edge of the ulcer may show the absence of either a part of or the entire posterior duodenal wall to the edge of the ulcer and the surface of the pancreas. In all such situations, if the anterior duodenal wall is sufficiently pliable, the closure can be accomplished in a simple manner without dissecting the duodenum from pancreatic tissue. This can be done with little fear of subsequent local complications.

Nissen[4] has described a method of duodenal closure for this type of ulcer, utilizing both the proximal and distal edges of the ulcer by suturing the edges of the ulcer to the anterior wall of the duodenum in two layers with interrupted sutures. He employed a third layer of interrupted sutures through the capsule of the pancreas and the duodenum. With this method the anterior wall of the duodenum rested on the base of the ulcer. Some authors[5–7] have

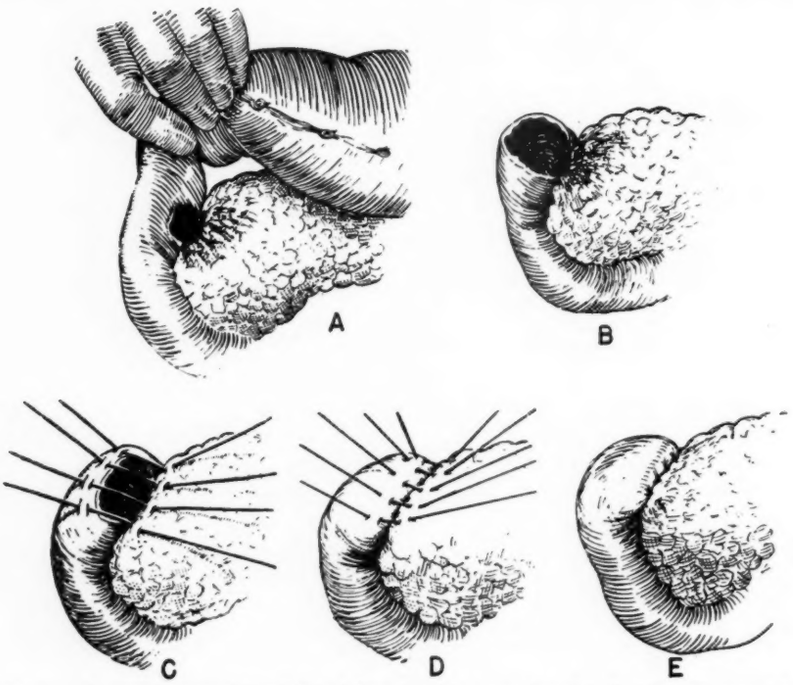

FIG. 1. A, demonstration of the defect in the posterior duodenal wall created by penetration of the dissection through the duodenal wall at the proximal edge of the ulcer. B, transected duodenum and the absence of part of the posterior duodenal wall to the edge of the ulcer shown. C, first layer of sutures. D, second layer of sutures. E, completed closure of duodenal stump.

reported various modifications of this type of closure. In those cases in which a pancreatic duct entered the base of the ulcer, Nissen recommended leaving the base of the ulcer uncovered by omitting the first row of sutures. The method to be described does not utilize the distal edge, and usually omits the placing of sutures through the proximal edge of the ulcer. The technic is relatively simple and can be performed fairly rapidly and safely, and with a minimum of dissection.

In the surgical approach to the posterior wall duodenal ulcer, the dissection of the duodenum is proceeded with in the usual manner. It is carried distal to the gastroepiploic vessels and the right gastroepiploic vessels are ligated and cut. The right gastric vessels are also clamped and cut. The position of the ulcer and the condition of the duodenum are carefully evaluated. If the position and size of the ulcer does not allow an ample and satisfactory posterior duodenal wall distal to it for the adequate standard open closure, the best method of duodenal closure is then determined. If the duodenal wall posteriorly is pliable and in satisfactory condition to hold sutures of silk, even though it is shortened, it may be utilized in the closure which will be accomplished either with two layers of Lembert or mattress sutures of silk. If the posterior wall is friable, edematous or excessively thickened, the posterior dissection is carried down to the ulcer, and the posterior wall removed to the edge of the ulcer. (Figs. 1A and B.) A satisfactory method is to continue bluntly freeing of the duodenum from the pancreas until the ulcer is broken into intentionally. The posterior duodenal wall bordering the ulcer edge laterally is trimmed as indicated, and is preserved if it is in suitable condition for closure purposes. The anterior wall of the duodenum is trimmed adequately so that a redundant wall will not interfere with the subsequent closure.

The following description refers to the duodenal closure totally omitting a part of or the entire posterior wall in completing the closure. The first row of interrupted Lembert sutures of No. 0000 silk are placed as shown in Figure 1C. There usually is no problem in placing these sutures near the edge of the ulcer, for as a rule there is some scarring in this area. The sutures should be placed superficially. The sutures on the duodenal side are placed through the seromuscular layers. After the first row of sutures

are tied, the second row is placed in a similar fashion. (Fig. 1D.) At this time one may be beyond the indurated area in the pancreas and it may be advisable to place the sutures transversely, taking two or three bites of small amounts of tissue and then completing the suture on the duodenal side in the Lembert fashion. There need be no worry about the deep structures in the pancreas or the pancreatic tissue itself if these sutures are placed in the manner described. After the second layer of sutures are tied (Fig. 1E), a third layer may be placed in a similar manner, or any neighboring fatty tissue used to cover the duodenal closure. Care must be taken to invert an adequate amount of duodenal wall at the ends of the closure. Adequate blood supply to the duodenal wall must be preserved, and the closure should be accomplished without undue tension.

Some variations of this method will of necessity be employed, depending on the position and size of the ulcer, or involvement of the upper or lower border of the duodenum at the site of transection. The general principles of the closure, however, remain unchanged. A specific factor which may interfere with the proper utilization of this method would be a thickened, scarred or edematous anterior wall which could not readily be inverted. In this situation, if an attempt has been made to close the duodenum and one is concerned, a small Foley catheter may be brought through the duodenal closure and the abdominal wall. The Foley bag is distended with 3 cc. of water, and the catheter can usually be removed safely in ten to twelve days.

In 216 duodenal closures following gastrectomy for duodenal ulcer performed at this hospital from July 1, 1949 to March 1, 1953, a two- or three-layer closure with interrupted silk sutures was employed in ninety-four patients. This method was used when the posterior duodenal wall was not considered adequate and safe for the application of a continuous catgut suture.

In approximately forty of these patients the posterior wall was either penetrated intentionally at the edge of the ulcer and the wall above the ulcer excised, or the remaining posterior wall above the ulcer appeared so friable that it was considered advisable to excise it down to the level of the pancreas to allow the closure to be accomplished without excessive tension. In this group the only complication of duodenal stump closure was one subphrenic abscess which responded promptly to drainage.

By utilizing the described method of duodenal closure, the posterior wall ulcer of the duodenum can be treated surgically in a safe and expeditious manner, and the number of gastroenterostomies, two-stage procedures and duodenal closures with catheter drainage, previously performed for this type of ulcer, can be considerably reduced.

SUMMARY

The problem of closure of the duodenal stump after partial gastrectomy for duodenal ulcer has been discussed. Emphasis, however, has been placed on the surgical treatment of the posterior wall ulcer of the first part of the duodenum, and a simplified approach to that problem presented. The method avoids unnecessary dissection which may traumatize the pancreas or common duct. The difficulties resulting from dissection in this area have been discussed by Millbourn,[8] Warren[9] and Dunphy.[10] Possible trauma to the pancreatic ducts or to the pancreatic blood supply have been commented upon by these authors. The primary advantage of the described method is its simplicity and effectiveness.

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Appendicitis in the Oriental Prisoner of War

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THE relative infrequency of acute appendicitis in Orientals has long been a debatable subject and very few references can be found in the American literature regarding the incidence, the nature of the disease, and the difficulties and pitfalls in diagnosis. The prospects of greater future contact with the Oriental race by young American physicians, particularly in military service, might well be anticipated, and warrants more thorough study and dissemination of all available material.

An opportunity to observe and record the manifestations of acute appendicitis as it occurred in a sizable group of Korean and Chinese men presented itself in the Prisoner of War Command in Korea during 1953. The period utilized for this study, January to June, 1953, was believed to be a relatively stable interval as regards the control of all prisoner activities, medical supervision, and availability of facilities and personnel for management of nontraumatic surgical problems. The 14th Field Hospital in Pusan was responsible for the hospital care and definitive surgical treatment of approximately 35,000 Korean and Chinese prisoners during the six-month period under consideration. It is notable that only thirteen cases of acute appendicitis were encountered. The difficulties of diagnosis, the virulence of the pathologic process, and the high incidence of complications (even in this small group) emphasized the need for greater attention to the manifestations of the disease in this type of patient. (Table 1.)

Diagnosis. The same general criteria ordinarily used in evaluating an acute condition within the abdomen were applied to this group of patients in an effort to establish a valid diagnosis and proper indications for surgical intervention. However, it was soon recognized that our prior knowledge and individual experience was somewhat limited in regard to three extremely commonplace diseases in the Oriental, namely, (1) tuberculous peritonitis, (2) intestinal worms and (3) amebiasis. As contact and experience with these diseases increased, it became more and more apparent that the preponderance of abdominal symptoms encountered in this group had origin in one of these three diseases, and indirectly focused attention on the infrequent case of acute appendicitis.

Tuberculous peritonitis was a constant consideration in the differential diagnosis of acute abdominal pain and was the most prevalent cause of abdominal symptoms in the large group of cases of pulmonary tuberculosis under observation in these prison compounds. The exact incidence of the disease in the over-all prison population is unknown. The presence of concomitant pulmonary, renal or bone tuberculosis in a patient presenting acute abdominal symptoms was always taken into consideration and definitely aided in the evaluation. However, three patients with acute appendicitis had active, moderate or far advanced pulmonary tuberculosis; one also had renal tuberculosis for which right nephrectomy had been performed two months prior to the attack of appendicitis. Tuberculous appendicitis as an individual entity[1] was not generally considered in the differential diagnosis, nor was there any suggestion of this disease from pathologic study of the surgical specimens.

Among the intestinal worms, Ascaris lumbricoides was the most common offender. Infestation with this parasite and the extremely high incidence of amebic colitis combined to produce abdominal symptoms of varying degree and severity in a high percentage of all Orientals. It is conceivable that abdominal pain and other gastrointestinal symptoms due to tuberculous peritonitis, worms or amebiasis are so commonplace in this group as to become tolerable and stoically acceptable. This situation would certainly explain some of the patients' procrastination in requesting medical care, and the high incidence of appendiceal perforation and abscess formation.

The absence of diarrhea was always a significant finding. Only two of the thirteen cases gave a definite history of multiple watery stools

TABLE I*

Case	Diarrhea	Pulmonary Tuberculosis	Acute Appendicitis	Acute Appendicitis with Local Peritonitis	Acute Appendicitis with Perforation, Abscess, Generalized Peritonitis	Postoperative Morbidity
I (K. Y. S.)					X	
II (C. B. H.)					X	
III (S. B. K.)		X		X		
IV (K. T. K.)					X	X
V (Y. D. S.)	X	X			X	X
VI (K. H. K.)	X				X	X
VII (K. Y. S.)			X			X
VIII (Y. K. C.)					X	
IX (S. J. H.)				X		X
X (K. S. T.)					X	X
XI (L. S. W.)			X			X
XII (J. T. J.)		X	X			X
XIII (K. T. B.)					X	X

* See text for description of pertinent data.

and both of these were unusually complicated problems. The only other notable diagnostic help was the ordinary routine white blood count and differential blood count. All cases of appendicitis had elevated leukocyte counts (9,000 to 20,700) with a high percentage of polymorphonuclear cells. The average case of tuberculous peritonitis, ascariasis or amebiasis presenting the clinical picture of acute appendicitis rarely was accompanied by the abnormal blood count characteristic of appendicitis. This corresponds to Miles' finding that significant leukocytosis was found in only 11.5 per cent of sixty-eight cases of intestinal parasites that mimicked surgical abdominal emergencies.[2] He pointed out the perplexing problem of differentiating acute appendicitis from acute cecal amebiasis and expressed the opinion that in doubtful cases the danger of postponing operation in acute appendicitis must be weighed against the risks of operating in intestinal amebiasis. Hence the simple blood count assumed increasing importance in the differential diagnosis.

Pathology. On the basis of the observed pathologic changes, acute appendicitis in the Oriental prisoner of war must be considered an unusually virulent process. Only three of the cases can be listed as uncomplicated acute appendicitis. Two of the remaining ten were classified as acute appendicitis with local peritonitis. The remaining eight cases were in the category of acute appendicitis with perforation, with or without abscess formation, or generalized peritonitis, or both. Since autopsies were performed on all patients who died, it was believed that no unrecognized cases escaped scrutiny.

Regarding etiology and pathogenesis, occasional references[3,4] have been made to (1) the influence of dietary factors, and (2) the Oriental manner of defecation, the squatting position, supposedly contributing to more complete cecal emptying. In regard to the first factor, few if any Western dietary innovations were introduced for over-all prisoner consumption. Rice was the basic food and made up part or all of every meal. In regard to the mode of defecation, no particular changes were introduced or observed.

As previously stated, the patients' voluntary or accustomed tolerance of abdominal pain contributed in some cases to delay in treatment and allowed the pathologic process to advance in an unrelenting fashion. One of the best examples of this problem was Case V, which typified the multiplicity of diseases encountered. A Korean man in his twenties gave a history of persistent abdominal pain of one month's duration which had increased in severity in the last three days, and was associated with anorexia, nausea, vomiting, fever and diarrhea. Examination revealed an acutely ill patient with generalized abdominal tenderness. The chest roentgenogram was interpreted as moderately advanced pulmonary tuberculosis and the abdominal film suggested massive intraperitoneal fluid. Many Ascaris ova were

found in the stool specimen and the leukocyte count was 15,150 with 76 per cent polymorphonuclear cells. A presumptive diagnosis of tuberculous peritonitis was made and the patient was observed for twenty-four hours. Abdominal distention developed and laparotomy revealed acute appendicitis with perforation, generalized peritonitis and multiple abscess formation. Appendectomy and abscess drainage were performed and despite postoperative ileus, left lower lobe atelectasis, hypoproteinemia, wound dehiscence and secondary closure, he eventually recovered and was discharged. One month later he was readmitted with acute purulent meningitis from which he died. This case was the only death in the group of cases under consideration.

Treatment. Appendectomy was performed in twelve of the thirteen cases. In Case VIII abscess drainage alone was the operative procedure. It should be added, however, that additional abscess drainage was performed in five of the patients in whom the appendix was removed.

Postoperative morbidity occurred in nine of the thirteen cases and included fever of 101°F. or higher, paralytic ileus, pelvic abscess, wound infection and dehiscence, pulmonary atelectasis, and diarrhea with demonstrable Ascaris or Endamoeba histolytica in the stools. The high morbidity rate reflects the magnitude of the entire problem. During the period of study good x-ray and laboratory facilities were available and utilized; blood, intravenous fluids, plasma and plasma substitutes, and antibiotics were all available in quantity and used as indicated.

The only death which occurred was in the case previously described; the patient recovered from appendectomy and multiple abscess drainage, was subsequently readmitted with meningitis, and died.

SUMMARY

Experience with acute appendicitis in the Oriental man is presented. The study is based on a small series of cases observed in a relatively well controlled population group under prisoner of war conditions in Korea. Attention is directed to the difficulties of differential diagnosis, considering particularly tuberculous peritonitis, intestinal worms and amebiasis.

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SURGICAL REVIEW

Primary Benign and Malignant Tumors of the Ureter

A REVIEW OF THE LITERATURE AND REPORT OF ONE BENIGN AND TWELVE MALIGNANT TUMORS

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TUMORS of the ureter may be divided into two main groups, namely, primary (benign or malignant) and secondary (metastatic). In this article discussion will be limited to the primary type which originates in the ureter and may be accompanied with secondary or metastatic tumors in the bladder.

The rapidly increasing number of primary ureteral tumors reported in the past twenty years indicates that the condition is no longer a rare clinical and pathologic entity. The serious and perplexing problems of diagnosis and treatment of this condition warrants the presentation of thirteen additional cases and a review of the literature.

The first presumptive case of primary neoplasm of the ureter was reported by Rayer,[192] who encountered a case in which there were widespread metastases at autopsy. The first authentic case of malignant neoplasm of the ureter to be confirmed by histologic study was a "medullary" carcinoma with metastases to the retroperitoneal lymph nodes reported by Wising and Blix.[213] The first case of primary malignant neoplasm in the English literature was described by Davy.[136] The first benign tumor was reported by Lebert.[169]

Incidence. Undoubtedly primary ureteral tumors are encountered more frequently than is indicated by the reports. In a combined series of 77,104 autopsies (i.e., Renner[193b] 13,854, Gilbert[150] 22,810, Bell[121] 37,000, Abeshouse[1] 3,440) there were seven primary ureteral tumors (one in 11,000).

In a combined series of 70,115 urologic hospital admissions (i.e., Colston[130] 22,000, Beacham[120] 34,215, Keen[32] 3,900, Abeshouse[1] 10,000) there were twenty primary ureteral tumors (one in 3,690).

Prior to 1900 only eight autopsy cases of primary malignant ureteral tumors were reported. In 1934 Scott[198] collected sixty-one cases and subsequently the condition was recognized more frequently, for Foord and Ferrier[146] gathered 145 cases, Scott[199] 182 cases, Kojen and Petkovic[164] 238 cases, and Mortensen and Murphy[186] 245 cases. I have collected 197 additional cases not included in or published subsequent to Mortensen and Murphy's article, and will add twelve personal cases, bringing the total of primary malignant ureteral tumors to 454 cases as of July 1, 1954.

Primary benign tumors are less common than the malignant type. In 1933 Joly[161] recorded only forty-nine cases. Subsequently, Johnson and Smith[160] collected fifty cases, Harding, Grant and Yates[153] fifty-seven cases, Kojen and Petkovic[164] sixty-one cases, and Mortesen and Murphy[186] seventy-seven cases. I have collected sixty cases not included in or published subsequent to Mortensen and Murphy's article, and will add one personal case, bringing the total of benign tumors to 138 cases. It should be pointed out that the small solitary papilloma has been classified as benign despite the fact that it is a potentially malignant tumor by virtue of its tendency to develop into a papillary carcinoma in untreated patients or to recur in a malignant form in treated patients.

Secondary or metastatic tumors of the ureter are rarer than both primary types. In 1952 Boyle and Brown[4] collected forty-two metastatic tumors, chiefly from intestinal and prostatic carcinoma. Since then six additional

cases have been reported. The author has observed five clinical cases and ten autopsy cases of metastatic tumors of the ureter which will be reported in another paper.

Unquestionably, many more cases of primary ureteral tumors have been encountered but have not been reported for various reasons. A few cases have been omitted intentionally because they had been reported in unavailable or obscure foreign journals.

The increased number of cases reported in the past twenty years may be attributed to several factors: (1) improved diagnostic technics; (2) greater awareness of the condition by urologists; (3) the incidence of ureteral neoplasm, like neoplasms elsewhere in the body, is on the increase. In recent years comparatively large series of malignant cases have been reported, i.e., Cook and Counsellor[133] eighteen cases, Tresidder and Warren[59] twelve cases, Baron and Green[3] eleven cases, Baker and Graf[2] thirteen cases, O'Conor,[44] Smith[55] and Harrison et al.[23] ten cases each.

Age. I have reviewed the distribution of primary ureteral tumors according to age of the patients. The greatest number of malignant cases, sixty-seven or 36 per cent, occured in the seventh decade, and the majority of cases (85 per cent) occurred in the sixth, seventh and eighth decades. The youngest patient in this group was twenty-four years of age (Roome et al.[51]), and the oldest patients were eighty (Zimmer[68] and Soloway[56]) and eighty-one (author's Case XII).

In the benign group the age incidence is definitely lower. The highest incidence was in the fourth and fifth decades, and the majority of cases (67 per cent) occurred in the fourth, fifth and sixth decades. The youngest patients in this group were ten years of age (Patch[106]) and eighteen (Pierce and Miner[108]), and the oldest was eighty (Levant[98]).

These statistics are in accord with those published by other authors.

Sex Distribution. Malignant ureteral tumors occur with greater frequency in the male (126 cases or 66 per cent) than in the female (sixty-two cases or 30 per cent); sex was not given in twenty-one cases. The benign group also showed a preponderance of males but the ratio of sex distribution was not as marked, i.e., thirty-three males (53 per cent) and twenty-eight females (47 per cent). There was no appreciable difference in the sex distribution in the various pathologic types of tumors.

Location and Number. The most common site for the benign and malignant tumors is the lower third of the ureter (74 per cent). The incidence decreases as the ureter is ascended. Multiple tumors are relatively rare in both types. The site of the primary malignant tumor in 209 malignant cases was the lower third in 145, middle third in twenty-six, upper third in eighteen, entire ureter in six and not stated fourteen. In the sixty-one benign cases the site was the lower third in thirty-six, middle in twelve, lower in twelve, and lower and middle in one. In the 209 malignant cases the tumor was single in 174 cases and multiple in twenty-two cases; the number was not stated in thirteen. In sixty-one benign cases there were fifty-seven single tumors and four multiple.

Side. The incidence of primary ureteral tumors is slightly higher in the left ureter than in the right. In the malignant group the right side was involved in ninety-three cases, the left in ninety-seven cases and both sides in four cases; the side was not given in fifteen cases. However, in the benign group the right side was involved in twenty-four cases, the left in thirty-four cases and both sides in one case; the side was not stated in two cases. These findings are in accord with those reported by other authors.

Pathology. In the cases reviewed by the author twenty-six types of malignant and thirteen types of benign tumors were reported. In the series of 209 cases the most common tumor was papillary (transitional cell) carcinoma (153 cases or 72 per cent); there were twenty-four cases of squamous cell carcinoma, twenty-one carcinoma undifferentiated, five adenocarcinoma and six sarcoma. The sarcomatous cases included two instances of leiomyosarcoma, two neurogenic sarcoma and one round cell sarcoma. Papillary carcinoma and squamous cell carcinoma comprised 84 per cent of the total cases.

In the sixty-one benign cases the pathologic varieties were thirty benign papillomas, seventeen fibromas or polyps, six vascular tumors (i.e., three hemangiomas, two angiomas and one lymphangioma), five granulomas and three endometriomas.

It is interesting to note that a papillary carcinoma developed in the ureteral stump following nephrectomy in eleven cases, (Baker

and Graf[2] two cases, Harrison et al.[23] two cases, Smith[55] two cases, Hamm and Lavalle[22] one case, Champion et al.[9] one, Palocz[45] one, Douglas[11] one, and Carroll[7] one); squamous cell carcinoma developed in two cases (Loef and Casella,[37] Abeshouse[1]), and benign papilloma in two cases (Bufalini,[78] Robecchi[110]). The interval of time elapsing after nephrectomy for non-malignant lesions and the development of the ureteral neoplasm varied from four months (Smith,[55] Hamm and Lavalle[22]) to fourteen years (Leof and Casella[37]) to twenty-five years (Baker and Graf[2]). A papillary carcinoma was observed in the left ureter two and a half years following a right nephrectomy in the case reported by Howet and LeClerc-Dandoy.[26] Marshall and Leary,[38] and James and Warren[29] found a papillary carcinoma in the ureter leading to the upper half of a right (double) and left fused supernumerary kidney, respectively. The aforementioned squamous cell carcinoma observed by Abeshouse[1] also occurred in the ureter leading to the upper segment of a left double kidney.

Size. The size and shape of primary ureteral tumors show a considerable variation depending upon the pathologic type and the duration of the growth. The benign tumors of urothelial origin, i.e., papillomas, are usually single small round masses varying from 2 to 10 mm. in diameter and attached to the ureteral wall by a thin narrow stalk or pedicle, but occasionally they are sessile and multiple. The characteristic histologic features of this type of tumor are: (1) orderly arrangement of the transitional cells in multiple layers imparting a palisade architecture of the tumor; (2) delicate connective tissue with one or more capillaries in the stroma of each villous component; (3) uniformity in the size, shape and polarity of the cellular components of each unit; (4) absence of mitotic figures; and (5) absence of invasion of basement membrane and submucosa. The non-epithelial benign tumors usually occur singly and seldom exceed the size of a grape. The pedunculated fibroma or vascular polyp is also attached by a narrow stalk and does not invade the deeper ureteral layers. The predominating elements are the fibrous connective tissue stroma in the former and the vascular elements in the latter. The granulomatous tumors may be somewhat larger in size (10 to 20 mm. in length) and may spread into or involve the deeper layers of the ureter. This type of tumor is regarded by many authors as being due to chronic inflammation, and the actual tumor areas are composed of hyperplastic and inflamed mucosa and submucosa.

The malignant tumors are larger and more extensive than the benign tumors. With the passing of time they spread through all layers of the ureteral wall and may invade the adjacent structures or organs. Harrison et al.[23] observed a case of papillary carcinoma involving the appendix. The papillary carcinomas are usually oval (2 to 3 mm. in diameter), sausage-shaped or fusiform (2 to 10 cm. in length) and often completely occlude the ureteral lumen. In an occasional case they may attain the size of a hen's egg (Tuffill[60]) or a duck's egg (Roome et al.[51]). They are usually single and rarely multiple except in the case of papillomatosis. These papillary tumors are firm and indurated in consistency. The histologic features are irregularity of the size and shape of the cells, mitotic figures, invasion of all layers and extension into the vascular and perineural lymphatics.

The squamous cell carcinomas are more insidious in their development and may attain the size of a large navel orange, as in one of the author's cases. This type of tumor quickly penetrates all layers of the ureters and invades the adjacent structures, and consequently are inoperable when the diagnosis is made at operation. The consistency is firmer than in the papillary type and may feel cartilaginous, as in Baker's case. The histologic features are characteristic of epithelioma, i.e., irregular size and shape of cells, "epithelial pearls" and "whorls," and invasion of all layers. These tumors metastasize early and extensively.

The sarcomas are large, rapid-growing tumors which invade the adjacent structures and metastasize early.

The following is a simple anatomic and histologic classification that applies equally well to tumors of the renal pelvis, ureter and bladder.

Benign tumors:

1. Epithelial (urothelial)
   (a) papilloma—papillary (pedunculated; sessile)
2. Connective tissue
   (a) polyp
   (b) fibroma
3. Vascular tissue
   (a) hemangioma
   (b) lymphangioma

4. Muscle tissue
   (a) myoma
5. Miscellaneous
   (a) granuloma
   (b) endometrioma, etc.

Malignant tumors:

1. Epithelial (urothelial)
   (a) Transitional cell—papillary, non-papillary
   (b) squamous cell (epidermoid carcinoma)
   (c) adenocarcinoma (hyperplasia)
   (d) undifferientiated epithelial types
2. Connective tissue
   (a) sarcoma
3. Vascular tissue
   (a) endothelioma
   (b) angiosarcoma, etc.
4. Muscle tissue
   (a) leiomyosarcoma
5. Miscellaneous
   (a) bilharzial carcinoma
   (b) neurogenic sarcoma, etc.

The behavior pattern of primary epithelial tumors of the ureter exhibits a striking similarity to epithelial tumors of the renal pelvis and bladder. The salient feature of these tumors is their active or latent tendency toward rapid propagation or multiplication which may manifest itself either as: (1) a recurrence, (2) a new tumor formation following complete removal of the primary tumor, and/or (3) the simultaneous or consecutive occurrence of multiple tumors in other parts of the urinary tract. In view of the common embryologic, pathologic and clinical features of epithelial tumors of the upper urinary tract, Melicow[183] has proposed the following terms: "urothelium" for the epithelial lining of the renal pelvis, ureter and bladder, and "urothelioma" for epithelial tumors of these organs. This concept permits a clearer interpretation of the new pathologic, physiologic, biologic and genetic findings accruing from the recent studies on experimental carcinogenesis.

ETIOLOGY

Although the exact cause of primary ureteral tumors has not been definitely established, several interesting theories have been proposed and a wide variety of real or apparent factors have been impugned, namely, (1) obstruction, (2) trauma or irritation, (3) infection, (4) hyperplastic epithelial changes, (5) specific exogenous or endogenous carcinogens, and (6) hormonal imbalance.

Obstruction. There is no absolute confirmatory evidence of a clinical or experimental nature to support the contention that obstruction of the urinary outflow *per se* serves as an etiologic factor in the production of urothelial tumors. In the past urologists and pathologists have repeatedly stated that primary spontaneous neoplasms of the urothelium are infrequently associated with the common obstructive lesions of the upper urinary tract, namely, ureteral stones, strictures and kinks, bladder stones, prostatic obstruction, etc. This statement is not consistent with the fact that primary spontaneous urothelial neoplasms of the bladder and ureter are more commonly encountered in men and women past the age of fifty when obstructive lesions of the vesical neck are most frequently observed.

In my opinion, the location of primary urothelial tumors of the renal pelvis, ureter and bladder is definitely influenced in some measure by the hydrostatics and mechanism of urination. In humans, who spend the greatest part of the day in the upright position, the most common site for the spontaneous and occupational type of urothelioma is the most dependent portion of the bladder, i.e., on the posterior wall or trigone near the ureteral and urethral orifices. Primary ureteral neoplasms occur most frequently in the lower third of the ureter, the lumen of which in the terminal portion has the smallest caliber and is the most favorable site for primary or secondary obstructive changes. In a study of 800 primary urothelial tumors of the upper urinary tract Masina[176] found forty cases (5 per cent) of primary ureteral tumors situated in the terminal portion (the final inch) of the ureter, i.e., juxtavesical, intramural and submucosal.

In quadruped animals (dogs, rabbits and mice) used in experimental carcinogenic studies it has been found that the urothelial tumor occurs most frequently on the anterior wall of the bladder. The relatively infrequent occurrence of urothelial tumors of the renal pelvis and ureters in man and animal may be explained by the fact that under normal circumstances the urine outflow from the renal tubules is unimpeded and constant in its passage down the pelvis and ureter into the bladder, where the urogenous carcinogenic agent is permitted to act upon the urothelial surface of the bladder

for variable periods of time or until micturition ensues. An acquired obstruction at any point in the upper urinary tract would permit longer contact with urothelial surface above the site of the obstruction.

Trauma. Trauma or irritation cannot be indicated as an etiologic factor in the development of urothelial tumors. This statement is supported by the fact that urothelial tumors of the renal pelvis, ureter and bladder are infrequently associated with primary or secondary calculi which ordinarily would serve as ideal irritating agents. The calculi present in cases of primary ureteral tumors were mainly of the secondary type and were presumably the result of urinary stasis and infection caused by the tumor.

Infection. It is generally agreed that the usual acute pyogenic type of urinary infection plays little or no role in the development of urothelial tumors. In the majority of primary ureteral tumors reviewed by the authors, urinary infection was absent except in the late stages of the disease in a few instances. Furthermore, it should be emphasized that there is no concrete evidence to show that specific carcinogens are derived from bacterial sources. The role of virus infection in the formation of urothelial tumors has likewise not been definitely established.

However, there is positive evidence to show that certain parasites may be responsible for tumors of the bladder in man and animals. For many years it has been known that bilharzial infection may produce tumors of the bladder. In a recent study of thirty-two cases of bilharzial infection of the urinary tract, Makar[174] observed two cases of bilharzial carcinoma of the ureter and several cases of bilharzial papillomas of the ureter. He described two types of bilharzial lesions in the ureter: (1) hyperplastic, i.e., the presence of nodules or papilloma-like bodies in the ureteral epithelium and underlying connective tissue; (2) hypoplastic, i.e., fibrosis of the epithelial lesions with calcification of the bilharzial ova giving rise to atrophic "sandy patches." Malignant carcinomatous changes in the hyperplastic lesions is a rare phenomenon.

Infectious papillomatosis of the rat's bladder associated with infestation of the urothelium of renal pelvis, ureter and bladder has been produced by a worm (trichosomoides crassicandans).

On the other hand, some authors have maintained that chronic urinary infection may serve as a predisposing or initiating factor in the formation of urothelial tumors by altering the tumor susceptibility of the urothelium. The effect of chronic infection upon the urothelium may be manifested either as hyperplastic epithelial changes or more rarely as infectious granulomas. Chronic infection has been regarded as the responsible factor in the rare cases of carcinoma of the bladder, superimposed upon chronic interstitial cystitis. Fagerstrom[142] maintained that the neoplastic epithelial changes in ureteral tumors are the result of local irritation from chronic infection or noxious agents in the body fluids and that epithelial buds and crypts are the bizarre expressions of epithelial hyperplasia caused by chronic infection.

The relationship of chronic infection to leukoplakia and squamous cell carcinoma of the renal pelvis and bladder has been stressed by Patch,[190] Kretschmer,[166] Ash,[118] McCrea[179] and others. In such instances the epidermoid changes are the result of metaplasia of transitional cells into the stratified squamous variety and serve as precursors of the non-papillary or squamous cell carcinoma.

Although it is generally recognized that carcinoma may develop in tissues subjected to chronic infection or irritation, it appears that the actual transformation of urothelial cells into true or potentially malignant cells requires a specific factor of endogenous or exogenous origin but unidentified to date. This same mechanism is most likely the one involved in those rare cases of squamous cell carcinoma developing in the stump of the ureter many years following removal of the ipsilateral kidney for pathologic reasons other than a neoplasm, i.e., lithiasis, pyonephrosis, acute or chronic pyelonephritis, etc.

Hyperplastic Epithelial Changes. The role of cell nests, described by Limbeck (1887) and van Brunn (1893),[125] in the formation of urothelial tumors has gained almost universal acceptance. These cell nests or epithelial buds represent infoldings or pinched-off segments of the basal layer of the urothelium of the renal pelvis, ureter and bladder, and are intimately connected with the surface epithelium. Morse[185] found these changes in 86 per cent of 125 consecutive autopsies and observed that in 58 per cent of the former group the cell nests were ac-

companied with chronic inflammatory changes. In some cases the histologic changes were indistinguishable from early malignancy and were composed of immature or incompletely differentiated cells of the transitional type. Bothe[122] found similar changes in close proximity to primary epithelial tumors of the upper urinary tract. Fagerstrom[142] studied sections taken from the pelvis and ureter in 120 unselected autopsies in persons below the age of twenty years and failed to find any cell nests.

These cell nests are prone to undergo metaplastic changes, such as: (1) pyelitis, ureteritis or cystitis cystica; (2) pyelitis, ureteritis or cystitic glandularis, and (3) occasionally adenocarcinoma. Jacob and Mau[28] reported two cases with pelvic and ureteral metaplastic changes simulating the intestinal mucosal pattern, and in one case it was associated with an adenocarcinoma of the ureter. Everett and Wayburn[139] described a case with diffuse infiltration of the renal pelves and ureters by cell nests, and in one area they found a very early squamous cell carcinoma. These cell nests and other chronic proliferative lesions of the urothelium may ultimately lead to the development of malignant changes either as the result of evolutionary metaplastic changes, the response to specific stimuli (chronic infection, irritation, etc.) or the result of activation by endogenous or exogenous carcinogen. The presence of numerous hyperplastic or proliferative lesions with malignant potentialities in various parts of the urothelial tract is regarded by some investigators as confirmatory evidence of the multicentric origin of urothelial tumors.

Occupational Urothelial Tumors. In 1895 Rehn[193a] first described tumors of the bladder in dye workers. Similar cases now have been reported from practically every country manufacturing analine dyes. A wide variety of hydrocarbons have been investigated and some of these chemical compounds were found to be powerful bladder irritants rather than true carcinogens. Alpha- and beta naphthylamine were found to be true carcinogens. In 1938 Hueper[154] and associates were able to reproduce tumors of the bladder in dogs fed beta naphthylamine.

For a long time the *modus operandi* of the chemical carcinogenic agent in the occupational and chemical experimental types of tumor of the bladder has been a controversial subject. Two main theories have prevailed: (1) The urogenous theory, sponsored by Hueper,[155,156] Oppenheimer,[187] Engle[138] and others, assumed that the carcinogenic agent is excreted in the urine and acts directly upon the urothelial surface. (2) The hematogenous theory, advocated by Ferguson,[143] Gay[148] and others, assumed that the carcinogenic agent circulates in the blood stream and reaches the urothelium by way of the terminal capillaries in the submucosal layer of the urothelium. Scher combined the chief features of both theories and added the concept of selective reabsorption of the hematogenously excreted agent. Neither theory satisfactorily explains the long latent period of tumor formation in dye workers during exposure, the apparent immunity of many dye workers to tumor formation or the fact that urothelial tumors often develop long after exposure to the chemical carcinogens has ceased.

In 1937 Ferguson[143] proposed a novel method of testing validity of the theories; first diverting the urinary stream by ureterosigmoidostomy or by other methods and then administering the chemical carcinogenic agent to the experimental animal. For the past three years my associates and I have had such a study in progress in dogs.* We have utilized the aforementioned methods as well as others, such as cutaneous ureterostomies, unilateral and bilateral intubated ureterostomy of the ligated ureters, unilateral and bilateral ureterosigmoidostomy, isolated bladder pouches with or without associated cystostomy and transplantation of one or both ureters to an artificial bladder derived from ileum or sigmoid. The results obtained have not been published to date but are in close agreement with those published by Scott and Boyd[202] and McDonald and Lund.[180]

In December, 1953, Scott and Boyd[202] employed similar technics and obtained striking results. They did not observe any bladder neoplasm in those dogs with a substitute bladder (transplantation of ureters into an isolated loop of sigmoid exposed to the same carcinogenic agent over long periods of time), nor did they observe any neoplamss in the bowel in dogs with transplantation of the ureters into the sigmoid. They were able to produce for the first time early proliferative neoplastic lesions of the ureter and renal pelvis and primary carcinoma of the ureter following the administration of beta naphthylamine to dogs whose

* Abeshouse, B. S. and Scherlis, I. *Urol. & Cutan. Rev.*, 55: 1, 1951.

ureters were transplanted to the intact sigmoid and to an isolated loop of sigmoid. The proliferative lesions of ureter and renal pelvis were observed after thirty-two months of administration of the chemical carcinogen in a dog with ureteral stasis following right sigmoidostomy. In each of the two cases of primary carcinoma of the ureter the tumor appeared approximately thirty-one months following the implantation of the ureters into an isolated loop of sigmoid.

In May, 1954, McDonald and Lund[180] administered large doses (600 mg.) of beta naphthylamine daily to female dogs and male rabbits in whom the dome of the bladder was surgically transformed into a pouch isolated from contact with the urine. Later the urinary stream was diverted by transplanting the ureters to the vagina or to the isolated bladder pouch. The average induction time for a carcinoma developed within the bladder of the dogs was twelve and three-quarter months. These induced neoplasms were found in various portions of the bladder and not necessarily in the most dependent part of the bladder, i.e., the anterior wall, as stated by Hueper et al.[156] In none of the dogs did carcinoma develop in the epithelium of the isolated bladder pouch not in contact with the urine. The rabbits appeared to be resistant to beta naphthylamine in the dose and time selected, for no malignant changes were encountered but hyperplasia and metaplasia were observed. The authors described two new lesions for the first time, i.e., renal adenoma and early transitional cell carcinoma of the collecting duct epithelium of the kidneys in the dogs.

My experiments and those of Scott and Boyd,[202] and McDonald and Lund[180] definitely establish the fact that epithelial contact with the urine is an essential prerequisite in the formation of experimental tumors of the bladder in dogs and may be regarded as confirmation of the urogenous theory of vesical carcinogenesis.

Spontaneous (Non-occupational) Urothelial Tumors. The etiology of this type of tumor has not been clearly established. It is possible that a carcinogen may be present in the blood or urine in some unrecognized or unsuspected form as a result of: (1) some hormonal imbalance; (2) undetermined metabolic change in the liver, kidney, spleen or other organs of the body; or (3) a potential carcinogenic agent of endogenous or exogenous origin which may be activated by chemical changes in the urine. Gualtieri et al.[151] regarded the carcinogen as a chemical catalytic agent which may originate as a normal end product of metabolism, a by-product of a metabolic disturbance, a specific bacterial agent or a cellular mutative change. They maintained that the carcinogenic agent functions only when the cellular elements of the urothelium are "conditioned" or "sensitized" by special "influences" or factors of a local or general nature.

Since the majority of benign and malignant ureteral tumors are observed in patients between fifty and sixty years of age, it is possible that the development of these tumors is influenced in an unknown manner by some undetermined metabolic or hormonal changes which are more prone to occur in this period of life. However, if such were the case, one should expect to find bilateral urothelial tumors of the renal pelvis and ureter as frequently as the unilateral type. Scott and Boyd[202] have suggested the possibility of a general agent or a local physiologic functional change affecting one kidney and altering its filtration threshold in such a manner as to permit the excretion of the carcinogenic agent on only one side.

PATHOGENESIS

There is no unanimity of opinion concerning the exact mechanism involved in the spread of multiplication of urothelial tumors of the renal pelvis, ureter and bladder. In the urologic literature one finds the oft-repeated statement that the more histologically benign types of urothelial tumors exhibit a greater tendency toward multiplicity and implantation than do the more malignant (undifferentiated) cellular types of tumors. However, the studies of Doss, Vest and others do not support or lend credence to this contention.

Three main theories have been proposed to explain the multiplicity and recurrence of urothelial tumors, namely, implantation, lymphatic permeation and multicentric origin.

Implantation. This theory implies that viable tumor cells from the parent tumor are transported downward in the urinary tract by the urine or by peristaltic action and are deposited as graft implants on the urothelial lining. This theory, which was originally proposed by Albarran[116] in 1902 and later sup-

ported by Kimball and Ferris,[162] Mackenzie and Ratner,[173] Thomas and Regnier,[208] McAlpine,[177] and others, has been utilized to explain the fact that 20 per cent of papillary carcinomas of the renal pelvis are accompanied with similar lesions in the bladder. The frequent involvement of the lower ureter on the same side and its rare occurrence on the opposite side in such cases is offered as further support. Implantation in a retrograde manner, i.e., in the direction opposite to the flow of urine, has been suggested by Mortensen.[142]

Lymphatic Permeation. This mode of spread was proposed by Hunt[157] but has not gained popular acceptance for various reasons. From a pathologic standpoint, it is apparent that most proliferative urothelial tumors grow at the surface level and not from the submucosal or deeper levels. From an anatomic standpoint, it appears that direct or retrograde dissemination of tumor cells through the lymphatics is not likely to occur, as the lymphatic drainage of the kidney, ureter and bladder do not form a continuous system. The lymphatic drainage of the kidney, ureter and bladder is directed outward to the lymph nodes about the renal pedicle, abdominal aorta, the common iliac and its branches, respectively. Clinical and pathologic studies show that primary neoplasms of the bladder are rarely accompanied with secondary tumors of the renal pelvis or upper two-thirds of the ureter. Involvement of the lower adjacent portion of the ureter by tumors of the bladder is more likely the result of direct extension rather than lymphatic permeation.

Multicentric Origin. This theory, which was originally proposed by Ewing[141] and later advocated by Broders,[124] McDonald and Priestley,[181] Fagerstrom,[142] Bothe[122] and others, has been generally accepted by urologists and pathologists. This theory implies that several centers in the urothelium are affected by carcinogens and produce neoplastic cells which appear simultaneously or at separate intervals and may coalesce or remain discrete. The frequent occurrence of tumors of the bladder pre- and postoperatively, with the ureteral tumors in the series reviewed by this author, lends support to the multicentric origin of urothelial tumor.

ASSOCIATED PATHOLOGY

Primary benign and malignant tumors of the ureter are accompanied with a wide variety of pathologic lesions. (Table 1.) Many of these lesions have no intimate or causal relationship to the ureteral tumor. The most common associated lesion was hydronephrosis which was present in at least fourteen benign and 198 malignant cases and was the result of partial or complete ureteral obstruction caused by the tumor. One case of pyonephrosis and one of hematonephrosis were present in the benign group, and ten cases of pyonephrosis (eight of the calculous variety) were reported in the malignant group.

TABLE 1
ASSOCIATED PATHOLOGY

Pathologic Lesions	Benign Cases	Malignant Cases
Bladder:		
Papilloma	1	7
Papillary carcinoma	1	19
Papillary carcinoma in diverticulum	..	1
Polyp	..	2
Cystitis	1	2
Leukoplakia	..	1
Kidney:		
Acute pyelonephritis	1	2
Chronic pyelonephritis	2	4
Hydronephrosis	14	198
Pyonephrosis, calculous	1	10
Hematonephrosis	1	...
Papillary carcinoma of pelvis	..	2
Leukoplakia of pelvis	..	1
Cyst of kidney	..	2
Cystic adenoma	..	1
Leiomyosarcoma	..	1
Perirenal hematoma	1	...
Perinephritis	..	1
Tuberculosis	..	1
Renal calculi	1	8
Ureter:		
Ureteral calculi	2	3
Ureteral stricture	1	2
Prostate:		
Benign hypertrophy	1	15
Carcinoma	..	2
Prostatitis	1	...
Urethra:		
Caruncle	2	...
Papillitis	..	1
Miscellaneous:		
Spermatocele	..	1
Uterine fibroid	1	1
Uterine polyp	..	1
Carcinoma of uterus	..	1
Extrauterine pregnancy	1	...
Cystic adenoma of ovary	..	1
Cholelithiasis	..	1
Lymphoma of stomach	..	1
Carcinoma of ascending colon	..	1
Carcinoma of palate	..	1
Carcinoma of lungs	..	1
Tumor of breast (benign)	..	1
Multiple lipomas	..	1
Tuberculosis of lungs	..	2
Tuberculosis of hip	..	1
Paget's disease of bones	1	1
Diabetes	..	2
Cardiac disease	..	2
Glaucoma	..	1
Total	34	307

In the early series reported by Foord and Ferrier, Scott and others, renal and ureteral calculi were reported in 15 to 20 per cent of the cases. Cook and Counsellor observed three cases of ureteral calculi in eighteen personal cases. In my series calculi in the upper urinary tract were relatively rare. Ureteral calculi were found in two benign cases (Husch,[95] Saxe[111]) and three malignant cases (Abeshouse,[1] Wishard,[65] Keen[32]). In Glazier's[19] case squamous cell carcinoma was reported nine years following right ureterolithotomy and in Taylor's[58] case a stone was removed from the affected ureter by cystoscopic manipulation two years prior to removal of the neoplasm.

Renal calculi was reported in one benign case (Patch[106]) and eight malignant cases (Baker and Graf[2] three cases, Loef and Casella,[37] Boyle and Brown,[4] Jacob and Mau,[28] Weidner,[63] and Smith[55]).

A malignant neoplasm was found in other parts of the body in eleven malignant cases, i.e., papillary carcinoma of the renal pelvis (Douglas[11]), papillary adenocarcinoma of lower major calyx of right kidney (Douglas), renal leiomyosarcoma (Champion et al.[9]), renal cystic adenoma (Hamm and Lavalle[22]), carcinoma of prostate (Abeshouse, two cases), carcinoma of ascending colon (Kontoff and Hershman[34]), carcinoma of soft palate (Holmes[25]), lymphoma of stomach (Holmes[25]), squamous cell carcinoma of lung (Creecy[10]) and cystadenoma of ovary (Harrison et al.[23]). The exact significance of these multiple tumors is not clear. It should be noted that papillary carcinoma of the bladder was observed in one benign and nineteen malignant cases which must be regarded as secondary or metastatic tumors.

SYMPTOMATOLOGY

Benign and malignant ureteral tumors usually present the same clinical picture except for those malignant cases with obvious metastases. Clinical experience reveals that there is no symptom complex pathognomonic of either benign or malignant ureteral tumors. The three outstanding symptoms are hematuria, pain and tumor mass, each of which in itself is of no great diagnostic value. This triad of symptoms was present in only two benign cases and ten malignant cases. The chief complaint is rarely of the type associated with local spread or distant metastases. Duration of symptoms in both the benign and malignant ureteral tumors varied from one day to nine years, with an average of eight months in the benign tumors and 24.2 months in the malignant cases. The delay in establishing the correct diagnosis, particularly in the malignant cases, is undoubtedly responsible for the poor results obtained by radical surgery.

Hematuria. Hematuria is the most conspicious symptom and was present in forty-five benign cases (75 per cent) and 153 malignant cases (75 per cent). It was the chief complaint in forty benign cases and 127 malignant cases. Blood in the urine may vary considerably in amount, duration and form. The hematuria is usually painless unless it is accompanied with clot formation or casts which may obstruct the outflow of the urine from the ureter or urethra. The blood in urine is macroscopic in most instances, being microscopic in only four benign and eight malignant cases. In a few cases it was so profuse as to seriously threaten life. The hematuria is usually present throughout the entire act of urination and rarely is terminal except in those cases in which the tumor has protruded through the ureteral orifice. The duration of hematuria also varies considerably, being present for shorter intervals (weeks or months) in the benign type and for longer intervals (several years) in cases of low grade malignancy.

Pain. Pain is the next most prominent symptom and was present in thirty-seven benign cases (62 per cent) and seventy-eight malignant cases (36 per cent). It was the chief complaint in thirty benign and thirty-two malignant cases. The pain may vary in duration, location and intensity as well as with the type of tumor. The chief types of pain are (1) that which simulates ureteral calculus (dull, non-radiating or sharp colicky), and (2) the metastatic type. The former is the most common type and is usually a dull constant or intermittent pain in the loin or flank on the affected side due to the accompanying hydronephrosis resulting from the partial obstruction induced by the ureteral tumor. Next in frequency is the sharp or colicky pain due to acute ureteral obstruction or spasm caused by the tumor or ureteral blood casts. In the latter type the pain may radiate to the groin or testicle. The colicky type of pain was present in three benign and eleven malignant cases.

The metastatic type of pain is less commonly observed, more variable in location and

of graver prognostic significance. When the tumor invades the adjacent peritoneum or intra-abdominal organs, the pain is constant and localized to the involved area and is occasionally referred to the upper abdominal quadrant or epigastrium. When the tumor invades the retroperitoneal area and involves the psoas muscles and the overlying nerves, the pain may simulate sciatica and be referred down to thigh or legs. When the tumor extends into the pelvic region, the pain may be referred to the rectum, vagina or perineum. Bizarre symptoms may be present when the ureteral neoplasm perforates into the peritoneal cavity (Campbell[6]) or into the vagina (Felber[85]). In an occasional case (Hunt,[157] Creecy,[10] Hinz and Mohnke[24]) the chief symptoms were of the cerebral type (headaches, coma, vertigo, etc.) due to metastases to the brain which completely overshadowed the ureteral tumor, unsuspected and discovered only at autopsy.

When the primary ureteral tumor is accompanied with ureteral calculus, the diagnostic difficulties are enhanced.

Tumor. A palpable mass was present in only five benign cases (8 per cent) and twenty-two malignant cases (10 per cent). It was the chief complaint in only one benign and five malignant cases. In those cases diagnosed early no mass was found in the kidney or ureteral areas. In those cases diagnosed late the mass was an enlarged hydronephrotic kidney palpable in the flank or upper abdominal quadrant. In a few late cases the ureteral tumor was sufficiently enlarged and invasive to produce a localized mass in the lower or mid-portion of the ureter palpable in the lower abdominal quadrant on the affected side (Foord and Ferrier,[146] Frauboes,[15] Campbell,[6] Hamm and Lavalle,[22] Jacob and Mau,[28] Baron and Green[3]). In several cases the tumor in the lower third of the ureter was palpable vaginally (Israel,[180] Laport and Muret,[188] Creecy,[10] Weidner,[63] Smith,[55] Sanchez and Fernandes,[47] Abeshouse[1] (Case x), Baron and Green[3]) or rectally (Foord and Ferrier,[146] Creecy,[10] Wiggins-Davies and Elwood,[64] Howet and LcClerc-Dandoy,[26] Tresidder and Warren[59]).

Other Symptoms. Constitutional symptoms, i.e., general malaise, loss of strength and appetite, fever, anemia, etc., were frequently observed in patients with malignant tumors accompanied with extensive upper urinary tract disease or metastases. Urinary difficulties other than hematuria were commonly encountered in patients with urinary infection or obstructive lesions at the vesical orifice. In cases of metastases to the liver and bowel, jaundice, nausea, vomiting, abdominal distention, constipation, etc., were conspicuous.

TABLE II
DIAGNOSIS IN REPORTED CASES OF URETERAL TUMORS

	Benign Tumors	Malignant Tumors
I. Correct	38 (70%)	105 (75%)
II. Incorrect:	17 (30%)	53 (25%)
Stone in ureter	5	8
Hydronephrosis	4	14
Tumor of renal pelvis	4	
Pyonephrosis	5	2
Tumor of kidney	2	9
Calculous pyonephrosis	2	2
Tumor of bladder		4
Retroperitoneal tumor		2
Renal tuberculosis		2
Tumor of ascending colon		1
Ureteral obstruction		2
Blood clot		1
Ureteral stricture		6
III. Diagnosis not stated	6	51
Total	61 cases	209 cases

DIAGNOSIS

Preoperative Diagnosis. The first correct preoperative diagnosis was made in 1902 by Albarran,[116] who saw a tumor protruding through the ureteral orifice at cystoscopy. As a result of the recent refinements in urologic and radiologic diagnostic technics and a greater awareness of the condition on the part of urologists, the correct preoperative diagnosis is being made more frequently. Despite these improvements, however, there are many cases of ureteral tumors in which the diagnosis is not made either at cystoscopy or even at operation. Prior to 1950 the correct diagnosis was made in 35 to 50 per cent of the cases of ureteral tumors. In the series of ureteral tumors reviewed by the authors covering the past five years, the correct diagnosis was made in 70 per cent of the benign cases and 75 per cent of the malignant cases. (Table II.)

Although a careful history, complete physical examination, adequate laboratory studies and a complete urologic study are valuable in establishing a correct diagnosis, the three most important diagnostic measures are: (1) staining of urinary sediment, (2) cystoscopy and (3) retrograde pyelography.

Urinalysis. In addition to gross or microscopic hematuria, examination of the urine may also reveal other important findings. Pyuria was present in those patients with an accompanying infection in the upper urinary tract and was noted in only eight benign cases and eighteen malignant cases.

Microscopic examination of the urinary sediment may also provide other valuable information. Zirconi[214] found epithelial "pearls" in the urine and Richter[195] found mitotic cells. Fowler,[147] and Baron and Green[3] detected tumor cells in the urine without special staining methods. Recently, the Papanicolaou method of staining the urinary sediment has been successfully employed by Taylor[58] and Marshall and Leary,[38] and should prove a very useful diagnostic adjuvant in future cases. According to Mortensen and Murphy,[186] villous pieces of tumor have been found in the urine in several cases; and when such pieces are spread on glass slides and appropriately stained, the diagnosis of a papillary ureteral tumor can be made with relative ease.

Cystoscopy. Cystoscopy is an inestimable and indispensable diagnostic aid. One of the fundamental rules, which should be indelibly impressed upon the minds of every urologist and clinician, is the absolute necessity of performing cystoscopy immediately in every case of hematuria in order to determine the source of the bleeding. With the introduction of the new local, intravenous and inhalant anesthetic drugs, cystoscopy has become an innocuous and painless procedure which can be easily and quickly performed in the urologist's office. In this manner the urologist is enabled to detect whether the bleeding is coming from the bladder or from one ureteral orifice, and may postpone ureteral catherization and retrograde pyelography until the patient is admitted to the hospital. The cystoscopist should not be lulled into a false sense of security by the finding of a small tumor of the bladder but should investigate the upper urinary tract to exclude papillary tumor of the renal pelvis or ureter. Blood was observed escaping from the ureteral orifice in five benign cases and fifty-five malignant cases. In two benign and eight malignant cases clots were seen protruding from the orifice.

Ureteral catheterization also provides invaluable information. An obstruction to the upward passage of a ureteral catheter may occur with any type of ureteral tumor but is more commonly observed with the pedunculated papillary type of tumor. If the catheter becomes engaged in the tumor mass, the most gentle manipulation of the catheter may cause bleeding through or around the catheter (Chevassu-Mock's sign[128]). This was reported in one benign and six malignant cases. Occasionally one may succeed in manipulating a Blasucci spiral tip or plain whistle tip catheter past the obstructing tumor mass. Once the catheter is passed beyond the obstructing tumor, clear or faintly blood-tinged urine is obtained (Marion's sign[175]). The latter sign was reported in one benign and eight malignant cases.

The following cystoscopic findings may be regarded as positive signs of a ureteral tumor: (1) tumor tissue protruding through the ureteral orifice; (2) detection of tumor cells in urine from affected side by means of the Papanicolaou stain; (3) biopsy of protruding ureteral tumor tissue obtained by means of Lowsley grasping forceps or by resection with a plain or loop electrode; (4) biopsy of an intra-ureteral tumor obtained by suction through the tip of a catheter (Lich and Maurer[170]), by a Johnson wire basket or by a small grasping forceps (Roome et al.[51]).

The following cystoscopic findings are presumptive signs of ureteral tumor but are sufficiently important to warrant a tentative diagnosis and to demand further investigation: (1) frank blood or a blood clot escaping from the ureteral orifice; (2) intra-ureteral obstruction to the passage of a catheter; (3) increased ureteral bleeding when the catheter meets an obstruction or passes the obstruction (Chevassu and Mock[128]). (4) drainage of clear urine when catheter passes the obstruction (Marion[175]); (5) presence of a small papillary tumor in the bladder near the ureteral orifice which may be associated with a pelvic or ureteral tumor; (6) minor bladder changes which may be associated with a tumor of the lower ureter, i.e., elevation of intramural portion of bladder, gaping ureterovesical orifice, and congestion or edema about the ureteral orifice.

Roentgenographic Studies. A plain scout (K. U. B.) film should be taken in every case of suspected ureteral tumor. While such a film does not reveal positive evidence of a ureteral tumor, it may disclose marked enlargement of the kidney outline on the affected side due to

the accompanying hydronephrosis. This same film may show calculi to be present in the kidney or ureter on the same or opposite side. The presence of metastatic lesions in the lungs or bones may be detected in the plain film taken of these areas. In the author's case of benign tumor (Case XIII) the papilloma was covered with calcium salts and gave the impression that one was dealing with a radiopaque stone in the upper ureter.

Retrograde ureteropyelography is the most dependable single diagnostic aid in cases in which the ureteral tumor does not protrude into the bladder. When the ureteral catheter meets an impassable obstruction, none of the dye injected by the gravity or syringe method may pass beyond the obstruction. The ureteral catheter met an impassable obstruction in twenty-three benign and eighty-four malignant cases. No dye passed beyond the obstruction in four benign and twenty-eight malignant cases.

The most reliable urographic sign is that of ureteral obstruction, i.e., a narrowing or obliteration of the outline of the ureter at the point of obstruction and the accompanying filling defect. A filling defect was observed in thirty benign cases and in seventy-nine malignant cases.

The shape of the filling defect varies with the size and shape of the tumor. Senger and Furey[54] pointed out that infiltrating tumors of the ureter (i.e., infiltrating papillary and squamous cell carcinoma) usually produce an irregular diminution in the caliber of the ureteral lumen with dilatation of the ureter above the obstruction, whereas the solid or pedunculated papillary tumors produce either a ring-shaped or goblet-shaped defect with dilatation of the ureter above and occasionally below the obstruction. Dilatation of the ureter below the level of the obstruction is an infrequent observation and is considered to be the result of mechanical blockage by the tumor which descends with each peristaltic contraction of the ureter and thus acts as a ball-valve obstruction. The density of opaque medium occasionally overshadows the outline of the tumor. In an exceptional case of benign pedunculated fibroma or papilloma the smooth, regular outline of the tumor may be clearly visualized as reported by Johnson and Smith.[160]

It is imperative that a complete ureterogram be obtained in every suspected case of ureteral tumor. With the conventional whistle tip catheter it is often difficult to outline the lower end of the ureter. When the catheter becomes obstructed by the tumor and no dye passes beyond the obstruction, it is advisable to place the patient in the Trendelenburg position before repeating the injection. If this procedure fails to outline the ureter, the whistle tip catheter should be removed and replaced by a different type of catheter such as a Braasch acorn bulb, Woodruff metal bulb, Ravich metal bulb or a Foley cone tip catheter. When one of the latter types of catheters is inserted into or past the ureteral orifice, particularly in the case of a tumor in the lower third of the ureter, one is often successful in getting sufficient dye beyond the obstruction and obtaining a satisfactory outline of the filling defect and of the upper ureter and pelvis. Rusche and Bacon[197] have demonstrated a constant filling defect in serial ureterograms taken with the Moore serial apparatus while injecting slowly through the gradually withdrawn catheter.

When the conventional or spiral tip catheter is passed beyond the obstruction and/or a sufficient amount of dye is injected beyond the obstruction, the pyelogram usually shows marked stasis in the ureter, pelvis and calyces. Every case of unexplained hydronephrosis should be carefully rechecked for the presence of an unsuspected ureteral tumor. The most difficult cases of ureteral tumors to diagnose are those accompanied with little or no dilatation of the ureter.

Pneumo-ureteropyelograms have been utilized to demonstrate the filling defect and dilatation of ureter and pelvis by several German urologists. This procedure appears to us to be fraught with the danger of air embolism in the presence of a bleeding or traumatized tumor.

Excretory urography has proved to be of relatively little diagnostic value. In the majority of cases in which it was employed the films usually showed no absence of dye in the affected hydronephrotic kidney resulting from obstruction of the ureter by the tumor. The enlarged outline of a non-functioning hydronephrotic kidney should serve as a warning to the urologist to search for the cause of the obstruction. In a few cases there was only slight concentration of the dye within the kidney pelvis, which indicated a seriously impaired or poorly functioning kidney but gave no clue regarding the causative lesion. In a

very small number of cases hydronephrosis and hydro-ureter were demonstrated, and in an even smaller group the filling defect in the ureter was depicted by this procedure.

Differential Diagnosis. A benign or malignant ureteral tumor may easily be mistaken for any of the following conditions: (1) non-opaque ureteral calculus, (2) blood clot, (3) papillary carcinoma of the renal pelvis and (4) obstruction of the ureter by an inflammatory or neoplastic mass of intrinsic or extrinsic origin.

A non-opaque calculus in the ureter can usually be ruled out by employing a wax-tipped catheter to detect the calculus. The absence of an irregularity or a filling defect in the ureteral outline in the ureteropyelogram and the demonstration of a calculus by pneumo-ureteropyelogram are other useful differential signs. Scott has pointed out that the passage of a ureteral catheter imparts a greater degree of resiliency in the case of a tumor and a more definite feeling of obstruction in the case of a stone.

A blood clot or cast within the lumen of the ureter may present a difficult diagnostic problem. The passage of a catheter is less likely to be impeded by a blood clot. In the case of a blood clot, pyelography repeated in several days will usually show no persistent defect. The demonstration of the causative lesion in the pelvis or calyces usually establishes the diagnosis.

Papillary carcinoma of the renal pelvis is usually accompanied with definite alteration in the outline of the pelvis and some degree of caliectasis. This type of tumor may be accompanied with ureteral implants in about 25 to 30 per cent of the cases which greatly enhances the diagnostic difficulties.

Compression of the ureter by a unilateral inflammatory or neoplastic mass of extrinsic origin is even more rare than a primary or secondary ureteral tumor. The most common type of extrinsic compression is the metastatic retroperitoneal lymph nodes in cases of primary neoplasms of the large bowel, cervix and fundus uteri and testes. These cases are seldom accompanied with outstanding urinary complaints or symptoms. The chief complaint is usually vague abdominal pain or distress. Since the compression of the ureter by the secondary lymph nodes is slow and gradual, evidence of renal impairment may be absent or inconspicuous in the early intravenous or retrograde pyelograms. The filling defect in the ureter produced by these extrinsic masses is more likely to be regular in outline and elongated in shape. Infiltrating ureteral neoplasms are not infrequently diagnosed as strictures of the ureter. It is important for the urologist to remember that stricture of the ureter with accompanying hydronephrosis is rare in patients of the cancer age. For all intents and purposes, stricture of the lower third of the ureter in this group of patients should be regarded as malignant until proved otherwise.

TREATMENT

No condition in urology poses a more serious problem than the operative treatment of ureteral tumors, as evidenced by the wide variety of operations employed and the poor results obtained. In 1943 Scott[199] tabulated thirty-nine different operations or combinations of operations utilized in the treatment of malignant neoplasms of the ureter. In 1953 Senger and Furey[54] reviewed 130 cases of ureteral tumors and recorded the use of fifteen different operations. The types of operations employed in the series reviewed by the author is indicated in Table III.

The lack of uniformity in the selection of the operative procedure by different urologists may be attributed to the fact that the choice of operation is influenced by various factors: (1) the general condition of the patient; (2) the functional status of the affected and opposite kidney; (3) the duration, location and pathologic type of the tumor; and (4) the technical difficulties encountered at operation.

The best results are contingent upon early diagnosis and prompt treatment. In malignant tumors the operation of choice is complete nephroureterectomy plus removal of a cuff of the adjacent bladder. In cases of definitely proved benign tumors, especially the simple polyp (fibroma) and vascular tumor, conservative surgery has been successfully employed; however, in the majority of benign tumors of the papillary type complete nephroureterectomy has been employed. The advocates of radical surgery in benign tumors have repeatedly stressed the fact that the so-called benign papillary tumors of the ureter, like all papillary tumors of urothelium, were potentially malignant tumors and that it is seldom possible to determine the exact degree

TABLE III

OPERATIVE TREATMENT OF URETERAL TUMORS

Operation	Benign Tumors		Malignant Tumors		Remarks
	Cases	Deaths	Cases	Deaths	
Nephroureterectomy and partial cystectomy (1 stage)	9	..	47	3	Death due to (1) shock, (2) ileus, (3) coma
Nephroureterectomy and partial cystectomy (2 stage)	2	..	16*	..	* 1 patient had had 2 previous operations (local excision plus nephroureterectomy, partial)
Nephroureterectomy (complete) (1 stage)	25	..	66*	..	* 1 patient had fulguration of intramural ureter
Nephroureterectomy (complete) (2 stage)	2	..	5*	1	Death due to ileus and wound infection; * 3 patients had x-ray therapy postoperatively
Nephroureterectomy (complete) transperitoneal	..	..	3	..	
Nephroureterectomy (partial) extraperitoneal	10	..	20	1	Death due to shock and hemorrhage
Nephroureterectomy (partial) plus fulguration through cystotomy	..	..	1	..	
Ureterectomy	..	..	8	1	Death due to cardiac failure and renal insufficiency; previous nephrectomy 7 cases
Segmental resection	..	..	2	..	
Segmental resection plus reanastomosis	1	..	....	..	
Segmental resection plus reanastomosis in bladder	1	..	3*	1	Death due to shock; * ipsilateral, contralateral and bilateral, 1 case each
Segmental resection plus cutaneous ureterostomy	..	..	3	..	
Excision (ureterotomy)	5	..	4*	..	* 1 case bilateral (right excision of 2 tumors, left nephroureterectomy, complete)
Excision (cystotomy)	2	..	....	..	
Excision (ureterotomy plus cystotomy)	1	..	....	..	
Excision of stump	..	..	7	1	Death due to hemorrhage
Nephrostomy	..	..	2*	1	* Death due to shock and renal insufficiency, 1 case bilateral
Exploratory operation	..	..	3	..	
Type of operation not stated	..	..	1	..	
Multiple operations	..	..	3	..	
No operation	3	..	18	..	
Total	61	..	211	9	4.6% operative mortality

of cellular malignancy or the absence of malignant tendencies even after obtaining a biopsy specimen of the tumor at cystoscopy or by obtaining a frozen section at operation.

In recent years several outstanding urologists (Ewert,[140] Vest[211] and others) have argued that no hard and fast rule should be applied to operative treatment of benign ureteral tumors. They have maintained that some form of conservative treatment is indicated in carefully selected cases. In support of their stand they have pointed out the following: (1) Benign tumors of the ureter, especially papilloma and even papillary carcinoma, grade 1, rarely undergo malignant degeneration and seldom metastasize. (2) Cases of benign papillary tumors have been treated conservatively and followed up for one to eight years without local recurrence or reimplantation in other parts of the urothelial tract. (3) Conservative surgery is indicated in view of the bilaterality of some papillary tumors, the frequent association of ureteral tumors with primary papillary tumors of the renal pelvis and bladder, and the tendency of such primary tumors to recur in other parts of the upper urinary tract. (4) Since the basic cause of urothelial tumors is unknown or undetermined in most cases, it is possible that the contralateral ureter or renal pelvis may become involved following removal of the kidney on the affected side.

Other urologists (Burns,[126] Davis,[135] Foley[145] and others including the author) have maintained that the conservative surgery for benign papillary tumors should be reserved only for those cases in which the contralateral kidney is absent or shows serious impairment of function. Those urologists utilizing the conservative operative treatment should always be mindful of the hazards of the conservative operation, i.e., stricture formation, fistula formation, faulty healing, secondary ureterectasis and pyelectasia, recurrence or further spontaneous new growths, implantation of tumor cells at operation, etc. When the urologist has any doubt as to the exact cellular pattern of the ureteral tumor after cystoscopic biopsy or frozen section at operation, it would appear to be far wiser and more discreet to err on the side of safety and perform complete nephroureterectomy in cases of benign papillary tumors of the ureter.

The factors influencing the choice of conservative surgery are: (1) demonstration of good function in both kidneys and a relatively small filling defect on cystoscopy and pyelography, (2) positive histologic evidence of benignity of the tumor on biopsy at cystoscopy or operation, and (3) absence of invasion of the ureteral wall at operation.

The operation of complete nephroureterectomy with or without segmental resection of the adjacent bladder may be performed in one or two stages depending upon the condition of the patient, the operative findings and the technical difficulties. Three types of incision have been employed: (1) a combination of an extraperitoneal incision (midline or Gibson type) to expose the lower ureter and bladder and a curved loin incision (Israel or Young type) for the removal of the kidney; (2) a curved loin incision extending downward and forward to the suprapubic area; and (3) a long rectus or pararectus incision of the transperitoneal type (Harrison et al.,[23] Hubert,[27] Rademaker[48]).

The first method is to be preferred since it permits operation at the site of the disease (lower ureter) first and enables the surgeon to stop the operation at this point if necessary. Furthermore, in those cases in which a positive diagnosis has not been made preoperatively between a tumor or a non-opaque stone, this exposure enables the surgeon to establish the correct diagnosis and preserve the kidney if possible. In a few cases the procedure has been reversed. The kidney has been exposed or removed first because of an error in diagnosis (renal neoplasm, stone, hydronephrosis, pyonephrosis, pelvic tumor, idiopathic hematuria, etc.) only to have the bleeding recur a short time later and the true nature of the causative lesion, ureteral tumor, detected at a subsequent examination (Baron and Green,[3] Tresidder and Warren[59] and others). In several cases in which the kidney was removed first, the ureter was removed or torn off at or above the site of the tumor.

There is no unanimity of opinion as to the best method of dealing with the intramural portion of the ureter and adjacent bladder wall in cases of benign or malignant tumors. Some urologists believe that in the case of solid carcinoma (squamous cell carcinoma) situated at some distance from the ureterovesical orifice, i.e., upper or middle third of the ureter, it is not necessary to remove the intramural portion of the ureter and the adjacent bladder.

Many urologists do not concur with this view. However, this procedure may be advisable and feasible because in a large number of cases of solid tumors extension beyond the ureter is found at operation and total extirpation of the entire tumor area is well nigh impossible. To attempt to remove a cuff of the bladder in such cases only adds considerably to the morbidity and mortality of the operation.

Most authorities are agreed that in cases of papillary carcinoma of any portion of the ureter and/or pelvis the intramural portion of the ureter must be destroyed or removed with the surrounding portion of the bladder. The management of the intramural portion of the ureter depends upon the mobility of the bladder, the thickness of the bladder wall and the degree of involvement of the bladder. Among the various procedures advocated are: (1) utilizing the ureter as a tractor to pull up the bladder, then clamping, excising a portion of the bladder and sewing the bladder without directly opening it (Caulk 1937[127]); (2) opening the lower portion of the ureter and introducing a fulgurating electrode to destroy the intramural, ureteral and adjacent vesical mucosa (Colston 1934[131]); (3) modifying Colston's method by first placing a purse-string suture in the bladder around the ureter, then excising a cuff of the bladder followed by fulguration and inversion of the cut ends (Kirwin[163]); (4) extravesical dissection of the intramural portion of the ureter leaving a small opening in the bladder to be sutured (Patton[191]); (5) transurethral resection of the intramural portion of the ureter prior to a one-stage nephroureterectomy (McDonald, Sturtevant and Upchurch [188]); and (6) transvesical excision of a cuff of bladder through a cystotomy incision (Bowie and Bors[123]). If the papillary ureteral tumor possesses a broad base with infiltration of the adjacent bladder wall, segmental resection of the bladder including the intramural portion of the ureter is necessary and can best be performed by the methods of Caulk or Kirwin.

Pre- and postoperative irradiation by radium or roentgen rays has not proved very satisfactory. This type of therapy was employed in only nine cases of malignant neoplasms: preoperatively, Tuffill;[60] postoperatively, Baron and Green,[3] Harrison et al.,[23] Marshall and Leary,[38] Soloway,[56] Tresidder and Warren[59] (four cases). Hess devised an apparatus for applying radium directly up the ureter prior to operation; however, due to the technical difficulties and the uncertainty of the results the method has never gained popular favor. Implantation of radon seeds in the vesical wall about the ureteral orifice was advocated by Foord and Ferrier,[146] and Tresidder and Warren.[59] The latter authors advocate its use in inoperable cases. Theoretically, roentgen irradiation should prove of value because of the tendency of papillary carcinoma of the ureter to recur locally and in the bladder. Thus Cook and Counsellor[133] suggested that roentgen therapy be administered postoperatively to all patients. However, the clinical experience of others has shown that the method has little practical value.

Chwalla,[129] and later Sziberth,[206] advocated the postoperative use of female hormone to prevent or control local recurrence or distant metastases. The value of this therapeutic method is extremely doubtful, as its use in tumors of the bladder by Shivers[203] and others has not produced definite curative results but only equivocal palliative results.

A wide variety of conservative operations has been suggested for benign tumors of the ureter, as indicated in the following classification modified after Vest.[211]

I. Electrocoagulation
   (a) at ureterovesical orifice or intramural ureter cystoscopically[149] or suprapubic cystotomy[105]
   (b) in any portion of ureter through ureterotomy[2,11]
II. Excision of tumor
   (a) transvesically[4,25,64]
   (b) transvesical ligation of tumor[207]
   (c) ureterotomy[108]
III. Resection of tumor and wall of ureter
   (a) partial with simple repair[2,11,51]
   (b) complete (segmental resection) supplemented by:
      1. end-to-end anastomosis[106]
      2. reimplantation of ureter into the bladder,[17,60] bowel or skin,[2,119,144,209,210]
      3. nephrostomy[61]
      4. ligation of ureter

MORTALITY AND MORBIDITY

The operative treatment of any malignant ureteral tumor is always a formidable and hazardous task, as the majority of these pa-

TABLE IV

FOLLOW-UP STUDY OF 109 MALIGNANT TUMORS (EXCLUSIVE OF SEVEN IMMEDIATE OPERATIVE DEATHS)

Time after Operation	Alive and Well	Dead	Recurrence				Metastases								Dead from Other Causes
			Local (Ureter)	Bladder	Prostate	Urethra	Lung	Bones	Liver	Spleen	Retroperitoneal Nodes	Inguinal Nodes	General Metastases	Brain	
1–6 mo.	17	13	2	4	2	2	6	6	6	1	9	2	5	1	Cardiac failure
6–12 mo.	14	9	3	5	..	..	..	2	..	..	2	1	4	..	Pulmonary embolism (1)
12–18 mo.	10	9	2	5	..	..	1	..	3	..	2	2	2	..	.....
18–24 mo.	6	7	..	5	..	..	..	3	1	..	..	..	1	..	.....
2–3 yr.	6	4	1	1	..	..	2	1	1	..	1	..	..	..	.....
3–4 yr.	6	..	1	2	..	..	..	..	..	..	..	..	..	..	.....
4–5 yr.	2	..	..	1	..	..	..	..	..	..	..	..	..	..	.....
5–6 yr.	2	1	1	1	..	..	..	..	..	..	..	..	..	..	.....
6–7 yr.	1	..	..	1	..	..	..	..	..	..	..	..	..	..	.....
7–8 yr.	3	..	1	2	..	..	..	..	..	..	..	..	1	..	.....
8–9 yr.	2	..	2	1	..	..	..	..	..	..	..	..	1	..	.....
9 yr.	1	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
10 yr.	..	1	..	..	..	..	..	..	..	..	..	..	..	..	Cerebral accident
11 yr.	1	..	..	..	..	..	..	..	..	..	..	..	..	..	.....
13 yr.	..	1	..	..	..	..	..	..	..	..	..	..	..	..	Coronary attack
Total.	71	45	13	29	2	2	9	12	11	1	14	5	14	1	.....

tients are in the fifth, sixth or seventh decades and are suffering from degenerative changes in the heart, lungs, liver and kidneys. In 1939 Foord and Ferrier[146] reported that the operative mortality was 40 per cent in forty-four nephrectomies performed in one stage and only 5 per cent in twenty-two operations performed in two stages. In 1950 Mortensen and Murphy[186] reviewed eighty-one cases and found no operative mortalities in twenty-eight cases of carcinoma and thirteen benign cases in which treatment was by one-stage operation, and in seven cases of carcinoma and six benign cases in which the two-stage operation was employed. In the series of sixty-one benign tumors reviewed by the author there were no deaths in the fifty-seven operative cases, and in 207 malignant cases there were only nine deaths in 191 operations (4.7 per cent): three deaths followed combined nephroureterectomy and cystectomy, and one each followed two-stage nephroureterectomy and partial nephrectomy, ureterectomy and partial nephroureterectomy, local excision through a ureterectomy incision, resection of a segment plus reimplantation in bladder, and nephrostomy. Although the latter results indicate a truly remarkable improvement in the operative treatment of this condition, they also serve as a testimony of the recent refinements and efficiency in urologic diagnostic methods which permit early and accurate diagnosis, and of the benefits derived from modern anesthesia and supportive therapy in the pre- and postoperative period.

Notwithstanding the aforementioned improvements, nephroureterectomy still carries a high morbidity, for in those cases with sufficient data one finds frequent reference to postoperative local or urinary tract infection, operative shock and postoperative ileus as well as the infrequent mention of peritonitis, pneumonia and embolism. In two cases of malignant tumor (Harrison et al.,[23] Keen[32]) it was necessary to employ laminectomy and chordotomy; in one case prefrontal lobotomy was performed in an attempt to relieve the pain caused by metastatic lesions in the vertebrae.

In 1934 Scott[198] found only two patients with malignant tumors who were alive after five years; in 1943 only six patients in a series of 183 with similar tumors were living and well more than five years after operation. In 1939

Foord and Ferrier[146] reported 34 per cent of 100 surgically treated patients were dead after three months.

A follow-up study of 109 malignant operative cases by the author revealed that seventy-one patients were alive and thirty-eight were dead.

TABLE V
SURVIVAL IN 109 OPERATIVE CASES OF MALIGNANT TUMORS (EXCLUSIVE OF IMMEDIATE OPERATIVE DEATHS*)

Type of Operation	No. of Cases	Average Survival (mo.)
Nephroureterectomy and partial cystectomy (1 stage)	43	45.7
Nephroureterectomy and partial cystectomy (2 stage)	10	20.5
Nephroureterectomy, complete (1 stage)	25	9.6
Nephroureterectomy, complete (2 stage)	4	18
Nephroureterectomy, complete (transperitoneal)	2	9.5
Nephroureterectomy, complete	10	10.9
Nephroureterectomy, complete (2 stage)	2	27
Ureterectomy	5	6
Segmental resection	6	18.2
Excision (ureterotomy)	2	22

* Total number of cases followed up: 109 (57%); total number of cases not followed up: 82 (43%); average length of time for follow-up: 24.2 months.

In the group of living patients thirty-one were alive one year or less, sixteen patients two years or less, six patients three years or less, six patients four years or less, and two patients five years or less; only ten patients survived more than five years. In the group of thirty-eight patients who survived operation but eventually succumbed, thirty-five were dead before three years had elapsed; one (O'Conor[44]) died after ten years of a "cerebral accident" and one (Wishard[65]) died after thirteen years of a coronary attack. The best results from the standpoint of survival following the various operations are recorded in Table IV. The best results were obtained following the one-stage and two-stage combined nephroureterectomy and partial cystectomy. (Table v.)

In 1943 Scott[199] found that only six of 182 patients were living and well more than four years after operation. Riches, Griffiths and Thackray[194] studied fifteen patients with papillary tumors of the ureter (in a combined series of 2,314 tumors of the kidney and ureter) and found that eight patients died within one year; one patient each lived two, three and five years, respectively, two lived four years and only one lived six years but developed carcinoma of the rectum; one patient was not followed up. O'Conor[44] studied eight patients with primary papillary tumors of the ureter treated with complete nephroureterectomy; he reported six patients alive with no signs of recurrence eleven, nine, eight, seven, five and two years after operation, and two dead (one after ten years from a "stroke" and one after three years from metastatic tumors of the bladder, urinary infection and renal insufficiency). Vest[211] has reported excellent results in three cases of benign tumors treated by excision, i.e., patients living and well without recurrence after one and a half, three and a half, and two and a half years, respectively.

Several other good results following operative removal of a primary malignant tumor have been reported. In 1950 Crance and Knickerbocker[134] recorded the case of a sixty-seven year old woman who was alive and free of all evidences of recurrence twenty-five years after removal of primary papillary carcinoma of the right ureter. Colston[132] recorded a patient with a papillary carcinoma who lived fourteen years following operation and died without evidence of recurrence. Krafft[165] and Scott[201] observed patients with papillary carcinoma who were alive and well eleven and a half and ten years after operation, respectively. The author has under his care at the present time a sixty-nine year old man who is in perfect health, with no evidence of local recurrence twenty-two years after right nephroureterectomy for papillary carcinoma of the ureter performed by Dr. H. H. Young in 1932 and suprapubic prostatectomy for benign hyperplasia performed by the author in 1947.

Recurrence. The value of combined nephroureterectomy and partial cystectomy in the treatment of papillary carcinoma of the ureter and/or the renal pelvis is substantiated by the studies of Kimball and Ferris.[162] They studied seventy-four cases of papillary carcinoma of the renal pelvis associated with similar tumors in the ureter and bladder developing after the original tumor was diagnosed or treated by incomplete operation, and found that new or

secondary tumors developed in the ureter or bladder in forty-eight cases. In the latter group the ureteral orifices and adjacent bladder were involved in 48 per cent, the ureter and distal portion of the bladder in 36 per cent and the bladder alone in 14 per cent. The total known recurrence was 64 per cent. On the other hand, there were no recurrences in the group of twenty-four cases of papillary carcinoma of the renal pelvis and ureter treated with combined nephroureterectomy and partial cystectomy.

In the series of 109 malignant cases with follow-up studies reviewed by the author, recurrence of the tumor following operation was reported in the bladder in twenty-nine cases, ureter thirteen cases, posterior urethra two cases, prostate two cases and skin of lumbar incision for nephrectomy two cases. It is interesting to note that the incidence of recurrence was lowest in those treated with a one-stage combined nephroureterectomy and partial cystectomy, as there were only four cases of recurrence in the bladder. Following a two-stage combined operation there were eight cases of recurrences in the bladder. The greatest incidence of recurrence in the ureter and bladder was noted after combined nephrectomy and partial ureterectomy, i.e., ten ureteral recurrences and ten recurrences in the bladder.

Of the total series of 209 malignant cases, thirty-five patients gave a history of being treated for a papilloma or papillary carcinoma of the bladder prior to the operation for the ureteral carcinoma. In the total series of sixty-one benign tumors only one patient was treated for tumor of the bladder prior to the diagnosis and treatment of the ureteral tumor. There were no recurrences in the ureter or bladder in benign cases treated with conservative or radical operation, with the exception of one case recorded by Felber.[84] In the latter instance nephroureterectomy was performed for "benign" papilloma of the left ureter; eight years later a papilloma developed in the middle third of the opposite ureter (right) and was treated with local excision and fulguration; two years later (10 years after the first operation) a transitional cell papillary carcinoma (grade III) developed which involved the right half of the bladder and vagina and was treated with partial cystectomy and vaginectomy. Baron and Green[3] reported another unusual case (Case 7) in a fifty-three year old man with hematuria who underwent left nephrectomy presumably for a hypernephroma; however, a large so-called benign papilloma was found in the mid-portion of the left ureter. Six years later a tumor of the bladder which showed early malignant features was treated with suprapubic electrocoagulation. Three years later another tumor of the bladder was treated with cystoscopic fulguration. Several months later papillary carcinoma was found in the stump of the left ureter which was removed by ureterectomy and partial cystectomy. Two years later partial cystectomy was performed for two papillary carcinomas of the bladder. This was followed with total cystectomy (eleven and a half years after nephrectomy) and the patient died on the eighth postoperative day.

Metastases. Metastases from malignant tumors of the ureter occur relatively early when there is a delay in diagnosis on the part of the patient or physician, or when treatment is postponed or inadequate. The distribution of the metastases in forty-five autopsies in the series of 139 cases collected by Foord and Ferrier[146] were regional lymph nodes forty-six, liver twenty-two, lungs sixteen, kidneys thirteen, bones twelve, peritoneum eight, iliac vessels two, vagina one and brain one. In 108 cases reviewed by the author the distribution followed essentially the same pattern, namely, regional lymph nodes fourteen, bones twelve, liver eleven, lungs nine, brain two and spleen one. Generalized carcinomatosis was reported in fourteen cases and inguinal glandular involvement was noted in five cases. The distribution of metastases in the non-operative or autopsy cases reviewed by the author was essentially the same as in the operative group.

Prognosis. Although the results of surgical treatment have shown a remarkable improvement in mortality and morbidity, the results from a curative and longevity standpoint are rather disappointing. In many cases the follow-up data were too meager to permit definite conclusions.

A follow-up study was reported in only nineteen benign and in 109 malignant cases. (Table v.)

In general, the prognosis was better in those cases in which the ureteral neoplasm was accompanied with a moderate-sized, well functioning hydronephrosis than in those with a non-functioning pyohydronephrosis. It appears that in the former group the tumor was slow-

growing and less infiltrating, whereas in the latter group the tumor grew rapidly, invaded the ureteral walls, spread beyond the confines of the ureter and was more likely to be accompanied with infection in the affected kidney.

In order to make a correct and accurate evaluation of the results of conservative and radical surgery in the treatment of benign and malignant tumors, one should check the results five and ten years after operation.

CASE REPORTS

Malignant Ureteral Tumors

CASE I. M. L. (Hosp. No. G.U. 298), a seventy year old white man, was admitted to the Sinai Hospital, Baltimore, Maryland, on February 9, 1937, complaining of hematuria and pain in both lumbar regions of two months' duration. He had been in good health until two months before when symptoms of prostatism developed, with gross hematuria on several occasions and dull pain in both lumbar areas and in the suprapubic region. Physical examination was negative except for tenderness in the right lumbar area and benign hypertrophy of the prostate (second degree). The patient's general condition was poor. The urine showed albumin 1 plus, many red blood cells and pus cells. At cystoscopy (February 10, 1937) a small papillary tumor (½ cm. in diameter) above the right ureteral orifice and moderate intravesical enlargement of median and both lateral lobes of the prostate were found. The right ureteral orifice was indurated and elevated. Attempts to pass Nos. 5 and 6 F. ureteral catheters up the right ureter were unsuccessful. Excretory urography revealed an enlarged right kidney outline but no dye appeared in the right pelvis after thirty minutes. Diagnosis was: (1) malignant papilloma of bladder, (2) papillary carcinoma of right renal pelvis and/or ureter and (3) benign hypertrophy of prostate.

On March 3rd transurethral resection of the tumor of the bladder and the prostate was performed. The convalesence was slow due to a complicating infection of the upper urinary tract. In view of the patients' poor general condition it was decided to perform a two-stage nephroureterectomy and partial cystectomy, and on May 7th a right ureterectomy and partial cystectomy were performed. The posoperative course was stormy due to bronchopneumonia and cardiac failure, and the patient died on May 16th (ninth postoperative day). The pathologic report revealed: (1) papillary carcinoma (transitional cell of right lower ureter and (2) papillary carcinoma of bladder (secondary implant).

Comment. This case is of particular interest because the primary ureteral neoplasm was accompanied with two other lesions (a secondary neoplasm of the bladder and benign enlargement of the prostate), and any of these lesions could very well have been responsible for the hematuria. Another important feature was the choice and type of operative treatment which was considerably influenced by the patient's complaints and general condition as well as by the pathologic nature of the lesions. Since the major complaints were symptoms of prostatism and the patient's general physical condition was poor, it was decided to perform a combined transurethral resection for the tumor of the bladder and the prostatic enlargement first and at a later date a two-stage nephroureterectomy and partial cystectomy. Unfortunately the patient died from bronchopneumonia and cardiac failure on the ninth day following ureterectomy and partial cystectomy. This was the first of two operative deaths in the author's series of nine operative cases.

CASE II. J. S. (Hosp. No. 3899), a fifty-four year old white man, was admitted to the Sinai Hospital on April 15, 1945, complaining of hematuria of two years' duration. He had been in good health until two years before when painless hematuria developed, with passage of clots which recurred three times at intervals of six months. There was also dull, intermittent pain in the left loin. Symptoms of prostatism had been present for the past year, during which time he had lost 10 pounds. The physical examination was negative except for slight tenderness in the right lumbar area and benign hypertrophy of the prostate (second degree). The urine was negative for albumin but showed many red blood cells and pus cells. Cystoscopy on April 11, 1945, revealed intravesical enlargement of both lateral lobes of the prostate but no tumor was observed. A No. 6 F. catheter met an obstruction at the right ureteropelvic junction, and injection of 6 cc. of sterile water into this catheter reproduced the patient's pain. Stereoscopic retrograde ureteropyelograms (right) showed that the dye stopped at the ureteropelvic junction, and also revealed a filling defect in this area with an

FIG. 1. Case II. Retrograde ureteropyelogram showing irregular ovoid filling defect in upper third of right ureter due to papillary carcinoma. No dye passed beyond the obstructing tumor.

FIG. 2. Case II. Photograph of operative specimen. The arrow points to the papillary carcinoma in the upper third of the right ureter. The dilated pelvis and calyces are clearly shown.

irregular dilatation of the upper third of the right ureter. (Fig. 1.) No dye passed into the renal pelvis. The following diagnosis was made: (1) tumor in upper third of right ureter and (2) benign hypertrophy of the prostate (second degree).

On April 20th a right nephroureterectomy (partial) was performed and only 14 cm. of the ureter were removed. The postoperative course was uneventful and the patient was discharged from the hospital on April 29th. Subsequently he returned for examination in June, August and November, 1945, and January, 1946, and was in excellent health. He died suddenly of coronary thrombosis on February 7, 1947 (twenty-two months postoperatively). The pathologic diagnosis was: (1) papillary carcinoma (transitional cell) of the upper right ureter (Fig. 2), (2) right hydronephrosis, (3) chronic right ureteritis and (4) chronic right pyelonephritis.

Comment. This case presented no unusual problem in diagnosis as attested by the characteristic cystoscopic and pyelographic findings. Inasmuch as the tumor appeared to be sharply defined with a minimum degree of infiltration of the ureteral wall, it was decided to remove the kidney and 14 cm. of the ureter. Following the operation the patient was in good health without any evidence of local recurrence or metastases for twenty-two months, at which time he died suddenly of coronary thrombosis.

CASE III. W. G. (Hosp. No. 41291), a seventy year old white man, was first admitted to the Sinai Hospital on May 25, 1946, complaining of painless hematuria of two weeks' duration. He had been in good health until one year before when signs of progressive cardiac failure developed. On several occasions in the previous two weeks he had had painless hematuria with symptoms of prostatism. Examination disclosed arteriosclerotic heart disease, ankle edema and benign enlargement of the prostrate. These were no masses or tenderness in the abdominal or lumbar areas. The urine showed albumin 1 plus, many red blood cells and pus cells. A complete urologic study revealed: (1) right hydronephrosis, (2) left double kidney with incomplete duplication of left ureter (junction of ureters at the upper level of the sacrum), (3) acute and chronic pyelonephritis of left double kidney, and (4) benign hypertrophy of prostate. Conservative treatment

was advised and the patient was discharged on June 15th.

His second admission to the hospital was on September 23, 1946, because of recurrent hematuria, pain in the left lumbar area and weekness. A complete urologic study was repeated with essentially the same findings except for an obstruction and filling defect in the ureter to the upper left hydronephrotic segment of the double kidney. On September 27, 1946, a left nephroureterectomy (partial) was performed. Both ureters were removed above the level of bifurcation at the promontory of the sacrum. The pathologic report was: (1) left double kidney with acute and chronic pyelonephritis, (2) hydronephrosis of the upper supernumerary segment of the left double kidney, (3) chronic ureteritis, (4) stricture in ureter of the upper segment of the double kidney. After an uneventful convalescence the patient was discharged on December 1st.

His third admission to the hospital was on September 27, 1951, with the complaint of dull pain in the left lumbar area radiating to the left lower quadrant of the abdomen of six months' duration. There had been a marked loss of weight and strength. Cardiac symptoms and pronounced anemia were also present. Examination disclosed exquisite tenderness in the left upper quadrant and tenderness over all the lumbar vertebrae; however, no masses were palpated. Roentgenograms of lungs and vertebrae were negative for metastases. Cystoscopic examination revealed an enlarged prostate but no tumor of the bladder. Both ureteral orifices appeared normal. A No. 6 F. catheter met an impassable obstruction 10 cm. up in the left ureter. A left ureterogram showed a definite filling defect at the point of bifurcation of the ureters whose outline appeared to be somewhat irregular. Only a small amount of dye passed up into each ureter to the level of the previous operative ligation. A tentative diagnosis was made of (1) left ureteral tumor or (2) retroperitoneal tumor.

On November 16th an exploratory operation was performed through a left pararectus incision of the modified Gibson type and a round irregular mass of the size of a navel orange (about 12 cm. in diameter) was found at the site of the previously ligated ureters. The mass and the remaining portion of the ureters were removed down to the bladder which was not opened or resected. The pathologic examination showed the periureteral mass to be infiltrated by scirrhous carcinoma of the immature squamous cell type (squamous cell carcinoma). The mass involved and originated in one ureter. The convalesence was relatively smooth and the patient was discharged on January 6, 1952.

His fourth admission was on February 21, 1952, because of severe pain in the left loin, weakness, cachexia and cardiac failure. Examination disclosed a hard mass in the lower end of the first lumbar incision. A biopsy specimen of this mass showed metastatic squamous cell carcinoma. Roentgenograms of the chest, long bones and vertebrae were negative for metastases. The patient's course was progressively downhill and he died of cardiac failure on March 16th (four months after ureterectomy). Autopsy was refused. The diagnosis was squamous cell carcinoma of the stump of the left ureter with metastases to surrounding tissue and to the scar of the lumbar nephrectomy wound.

Comment. An interesting feature of this case is the fact that the obstruction and filling defect in the ureter leading to the upper segment of the left double kidney were recognized by the urologist and roentgenologist as being highly suggestive of a ureteral neoplasm but were regarded more likely as the result of an obstruction or stricture of the ureter caused by chronic infection. In view of the patient's poor cardiac status, conservative nephrectomy and partial ureterectomy were performed at the first operation. Approximately five years later he returned complaining of pain in the left lower quadrant, anorexia, loss of weight, etc. Urologic study revealed an impassable obstruction about 10 cm. up in the left ureter, and a diagnosis of ureteral or retroperitoneal tumor was made. Exploratory operation disclosed an infiltrating squamous cell carcinoma about the size of a large naval orange beginning in the stump of one of the double ureters and extending downward into the undivided portion of the ureter. Ureterectomy was carried out with some difficulty due to the infiltrating malignant process in the surrounding tissues. Subsequently, a metastatic squamous cell tumor developed in the lower end of the old nephrectomy wound.

One cannot state with absolute certainty whether the squamous cell carcinoma was present in the ureter at the time of the nephrectomy and was overlooked or whether it developed in the stump of the ureter at some time after the

nephrectomy. The sequence of events, the train of symptoms and the subsequent course strongly suggest the former as the more logical explanation.

Case iv. M. F. (Hosp. No. 42056), a seventy-two year old white man, was admitted to the Sinai Hospital on May 12, 1947, with the complaint of weakness and chronic non-productive cough of two weeks' duration. Previously he had been confined to this hospital from June 27 to November 16, 1946. At that time he complained of hematuria of eight months' duration, pain in both kidney regions and prostatism, and was found to have transitional cell papillary carcinoma of the bladder, a neoplasm or cyst of the right kidney, carcinoma of the lower left ureter, left hydronephrosis and benign hypertrophy of the prostate. Because of his poor condition and poor prognosis a minimum of conservative operative treatment was instituted. Nine months later suprapubic cystostomy with excision of the tumor of the bladder was performed and implantation of radon seeds was done. Three months later transurethral resection of the prostate was performed. Subsequently his course was progressively downhill. He lost considerable weight and strength and was confined to bed. In the two weeks prior to the present admission he became hoarse; he spoke with difficulty and had a non-productive cough. The urinary output was extremely low and he was troubled with marked urgency and frequency. At examination he appeared markedly emaciated and debilitated. He had a left exophthalmos with loss of vision in that eye. Many fine rales, diminished breath sounds and diminished percussion note were present over both lungs and a soft systolic murmur was heard over the heart. A soft round mass (about 10 cm. in diameter) protruded through the suprapubic incision and a firm oval mass (approximately 15 by 10 cm.) was palpable in the left flank. The prostate was enlarged to the second degree. Urine contained albumin 3 plus and was grossly bloody. He died in cardiac failure with pulmonary edema on May 13, 1947.

At autopsy the following lesions were found: (1) primary infiltrating papillary carcinoma of the left ureter (lower third), (2) transitional cell papillary carcinoma of the bladder and diverticulum of the bladder, (3) metastatic carcinoma in the perirenal and periaortic lymph nodes, (4) solitary cyst of right kidney, (5) coronary heart disease, (6) generalized arteriosclerosis, (7) pulmonary edema and congestion and hydrothorax, (8) cholelithiasis and (9) aberrant pancreas in duodenum.

Comment. This case emphasizes the danger of procrastination on the part of the patient, who failed to seek medical assistance despite gross hematuria of eight months' duration, bilateral renal pain and symptoms of prostatism. By the time he submitted to a complete urologic study he was found to be a veritable pathologic wreck harboring in addition to his arteriosclerotic cardiovascular disease a primary left ureteral neoplasm with hydronephrosis and hydro-ureter, a secondary bladder neoplasm, large benign hypertrophy of the prostate, and a malignant tumor or cyst in the right kidney. His poor physical condition permitted only the most conservative operative therapy; consequently, the carcinoma of the bladder was treated first by suprapubic excision, fulguration and implantation of radon seeds, and three months later transurethral resection of the prostate was performed. However, the neoplastic processes exacted a quick toll and the patient died of cardiac failure and pulmonary edema. Autopsy not only confirmed the foregoing diagnoses but also revealed a previously unsuspected secondary papillary carcinomatous tumor in a bladder diverticulum, a large solitary cyst in the right kidney and extensive metastatic involvement of the perirenal and periaortic lymph nodes.

Case v. F. R. (Hosp. No. 81220), a sixty-five year old white man, was first admitted to the Sinai Hospital on February 5, 1951, with the complaint of pain in the right flank, and pyuria of two months' duration. A tumor of the bladder had been removed by segmental resection twenty years previously and he had suffered from cardiac disease (angina) two years before this admission. In November, 1950, he was in another hospital for nineteen days with chills, fever, back pain, nausea, vomiting and pyuria. For the previous two months there had been recurring intermittent attacks of pain in the right flank, chills, fever, pyuria, weakness and symptoms of prostatism but no gross hematuria. Physical examination showed no abnormalities. Excretory urography revealed left hydronephrosis (second degree) and no function in the right kidney after thirty minutes. The urine was negative except for 5 to 10 pus cells per high power field. At cys-

toscopy on February 6th a papillary tumor was observed protruding from the right ureteral orifice. On the inter-ureteric ridge were two small papillary tumors; one was 0.5 cm. medial to the right orifice and the other was immediately adjacent to and covered the left ureter. A biopsy specimen was taken from the tumors on the inter-ureteric ridge, and all of the tumors of the bladder were removed with a resectoscope and were thoroughly fulgurated. The pathologic report indicated a benign papilloma of the bladder. No biopsy specimen was taken from the tumor protruding from the right orifice. A No. 6 F. catheter was passed up the left ureter with ease. Retrograde pyelography showed moderate left hydronephrosis. Cystoscopy was repeated on February 9th and a No. 5 F. catheter met an impassable obstruction within the right ureteral orifice and caused moderate bleeding around the catheter (Chevassu-Mock's sign). A No. 4 F. catheter met the same obstruction but after some manipulation was passed up into the pelvis and drained a large amount (30 cc.) of purulent urine within a few minutes (Marion's sign). An intravenous injection of 1 cc. indigo carmine was given and dye appeared from the left kidney in seven minutes in good concentration but no dye appeared from the right side in one hour. A retrograde right ureteropyelogram showed a very large right hydronephrosis with marked dilatation of the upper two-thirds of the ureter. An irregular filling defect involved the lower third of the right ureter. A tentative diagnosis was made of: (1) papillary carcinoma of the right ureter (lower third) with secondary tumor implantations in the bladder, and (2) pyonephrosis, right. Operation was advised but the patient requested postponement for several weeks to arrange his affairs. He was discharged on February 11th.

On his second admission on March 6th a one-stage operation (combined nephroureterectomy and partial cystectomy) was recommended. On March 9th the operation was limited to a right nephrectomy and partial ureterectomy because of pathologic changes encountered about the kidney, technical difficulties and the patient's poor condition during operation. The pathologic report disclosed: (1) acute and chronic pyelonephritis, (2) granulomatous pyonephrosis, and (3) tubular adenoma. The postoperative course was satisfactory. At operation on March 16th a right ureterectomy and partial cystectomy were performed. The pathologic diagnosis was a papillary carcinoma (transitional cell) of the lower third of the ureter. The immediate postoperative course was complicated by acute ileus, acute retention of urine, renal insufficiency and infection in both operative wounds. Despite supportive therapy, i.e., transfusions, antibiotics, etc., his course was progressively downhill and he died on June 26, 1951 (three months and ten days postoperatively) of chronic renal insufficiency, bronchopneumonia and cardiac failure. At autopsy the positive findings were: (1) acute suppurative pyelonephritis and pyonephrosis, left, (2) left hydroureter due to carcinomatous infiltration of the lower ureteral wall, (3) metastatic carcinoma involving right and left inguinal, periaortic, mesenteric, periportal, peripancreatic and tracheobronchial nodes, (4) acute cystitis, (5) bronchopneumonia (right upper lobe), pulmonary edema and congestion, (6) coronary heart disease (arteriosclerosis) with old myocardial infarct, (7) benign hypertrophy of the prostate, (8) chronic focal perihepatitis and perisplenitis, and (9) colloid adenomas of thyroid.

Comment. Although hematuria was not a presenting symptom in this case, the diagnosis of primary papillary carcinoma of the right ureter with metastases to the bladder was readily established by cystoscopy and retrograde pyelography. A combined one-stage nephroureterectomy and partial cystectomy was contemplated; however, due to technical difficulties and pathologic findings the first operation was limited to nephrectomy and ten days later ureterectomy and partial cystectomy were performed. The patient's postoperative course was stormy, being complicated by acute ileus, renal insufficiency, retention of urine and infection of the wound which led to fatal termination 100 days after the second operation.

Autopsy revealed the unusual finding of a metastatic carcinomatous infiltration of the lower left ureter with accompanying acute suppurative pyelonephritis and pyonephrosis which was responsible for the postoperative acute renal insufficiency.

CASE VI. M. L. (Hosp. No. 93296), a sixty year old white man, was admitted to the Sinai Hospital on June 15, 1952, with a complaint of painless hematuria of forty-three days'

duration. Physical examination showed no abnormalities. A plain film of the abdomen was negative except for a suspicious calcified shadow in the region of the upper third of the right ureter. At cystoscopy on June 17th blood was seen escaping from the right ureter; also seen were a papillomatous tumor (15 cm. in diameter) above the right ureteral orifice and a second pea-sized tumor near the vesical orifice. A No. 6 F. catheter met an obstruction in the upper third of the right ureter, which was overcome. Bilateral retrograde ureteropyelography showed a normal left kidney pelvis, calyces and ureter, and on the right side a filling defect was present in the upper right ureter with moderate dilatation of the renal pelvis and calyces. The bladder tumors were resected and fulgurated with a McCarthy resectoscope. It was believed that the filling defect in the right ureter was due to either a tumor, blood clot or stone.

On June 20th a right nephroureterectomy and partial cystectomy were performed and a stone was found in the upper third of the ureter. The patient had an uneventful recovery and was discharged on July 11, 1952. The pathologic report was: (1) papillary carcinoma (transitional cell) grade II of upper right ureter, (2) right ureteral stone, and (3) cortical scarring and intrarenal calcium deposits in right kidney.

The patient returned on March 20, 1953 (eight months postoperatively), with gross hematuria. A pea-sized papillary tumor was found in the bladder in the region of the scar and was fulgurated. On December 18th (seventeen months postoperatively) he had hematuria again and a small papillary tumor was found on the right side of the bladder and fulgurated. On July 1, 1954 (twenty-five months postoperatively), his condition was excellent with no further recurrence of bladder tumors.

Comment. This case presented no unusual problems in diagnosis or treatment. The striking features of this case were the presence of an accompanying ureteral stone and the recurrence of secondary tumors of the bladder eight and seventeen months postoperatively. This case also emphasizes the importance and value of systematic and periodic re-examination of every patient who has had a papillary tumor within the urothelial tract.

CASE VII. H. C. (Hosp. No. 85460), a forty-six year old white man, was first admitted to the Sinai Hospital on July 29, 1951, with the complaint of hematuria of ten days' duration and symptoms of prostatism for one year. The physical examination showed no abnormality except for benign enlargement of the prostate (first degree) and adenoma of the thyroid. At cystoscopy on July 30th a papillary tumor was found on the right side of the bladder above the right ureter. On July 31st segmental resection of the bladder was performed. The pathologic report was transitional cell papillary carcinoma of the bladder. The postoperative course was uneventful and the patient was discharged on August 9th.

He was again admitted on September 28, 1952, with hematuria of one week's duration. Examination was entirely negative. The urine showed innumerable red blood cells. At cystoscopy on September 29th blood was seen escaping from the left ureter. A No. 5 F. catheter met an impassable obstruction 12 cm. up in the left ureter but a No. 5 F. catheter passed up the right ureter to the pelvis with ease. Bilateral retrograde ureteropyelograms were taken and showed the right kidney pelvis, calyces and ureter to be normal. Several left ureteropyelograms were taken and a constant irregular filling defect was observed in the lower third of the left ureter with dilatation of the lower ureter below the obstruction and marked pyelectasis, caliectasis and ureterectasis above the obstruction. (Fig. 3.) The same findings were observed in the excretory urographic films. A diagnosis was made of malignant tumor of the left ureter. On November 5th a left nephroureterectomy and partial cystectomy were performed; the patient made an uneventful recovery and was discharged on November 20, 1952. The pathologic report was: (1) papillary carcinoma (transitional cell) of the left lower ureter, (2) chronic ureteritis, (3) hydronephrosis, left, and (4) focal scarring of left kidney.

The patient's third admission was on January 19, 1954. On the following day a subtotal thyroidectomy was performed for a nontoxic substernal nodular goiter. He made an uneventful recovery and was discharged on January 25, 1954. He returned for examination on July 1st (twenty-one months after nephroureterectomy and partial cystectomy) and was in excellent health with no urinary symptoms. The urine was normal, and physical examination and cystoscopy showed no abnormalities.

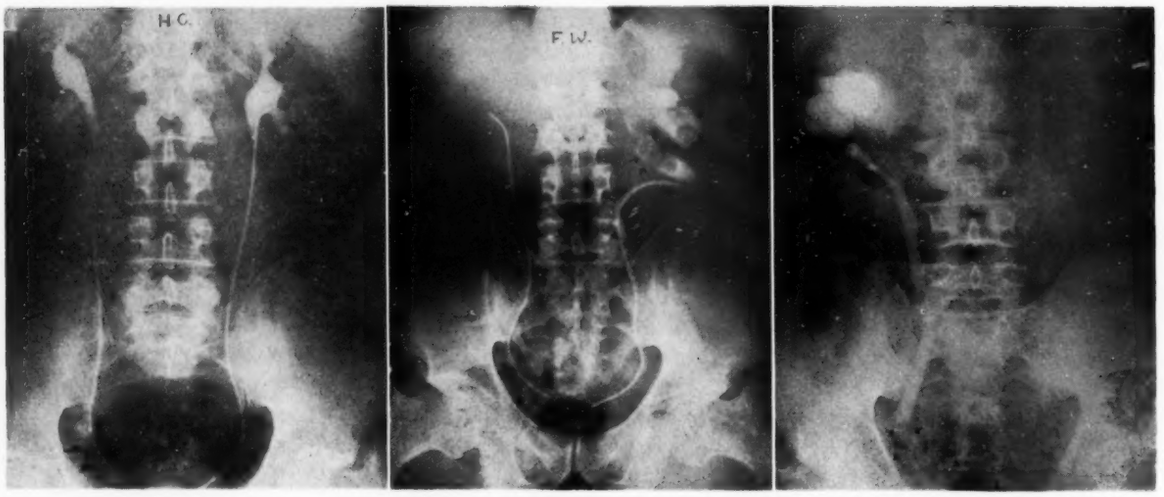

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FIG. 3. Case VII. Bilateral retrograde pyelogram shows a slightly dilated right pelvis and a normal left pelvis. The filling defect in the lower third of the right ureter is indicated by the arrows.

FIG. 4. Case VIII. Left retrograde pyelogram showing the catheter obstructed at the kink in the ureter immediately below the filling defect in the upper third of the ureter. A small amount of the dye passed into the dilated left renal pelvis and calyces.

FIG. 5. Case IX. Right ureteropyelogram showing a large irregular filling defect in the lower third of the ureter with marked pyelectasia, caliectasis and ureterectasis above the tumor area.

Comment. An interesting feature in this case was the presence of a small tumor of the bladder which was treated by segmental resection ten months prior to one-stage combined nephroureterectomy and partial cystectomy. The separate occurrence of tumor of the bladder and ureter is in accord with the theory of the multicentric origin of urothelial tumors.

CASE VIII. F. W. (Hosp. No. 106958), a fifty-one year old white man, was first admitted to the Sinai Hospital on August 3, 1950, with the complaint of back pain of two weeks' duration. He had been in good health until one and a half years before when he had symptoms of prostatism due to benign enlargement of the prostate and had been treated by transurethral resection of the prostate at another hospital. He had been told that the right kidney was enlarged on the roentgenograms. Six months before the present admission he had gross painless hematuria lasting four days, and three weeks before this admission severe back pain developed which was more marked in the right lumbar area. Physical examination revealed tenderness in the right lumbar area with no mass, bilateral inguinal hernias and pulmonary changes due to asthma. The urine contained albumin 1 plus, many pus cells but no red blood cells. Roentgenograms of the lungs, long bones and abdomen were negative. At cystoscopy on August 4, 1953, the bladder walls were negative for tumors. A No. 6 F. catheter was passed up each ureter to the pelvis with ease. Examination of the urine from the left kidney was negative, but an occasional pus cell was seen in the urine from the right kidney. An intravenous indigo carmine test showed a normal appearance and concentration from each kidney. Bilateral retrograde ureteropyelograms showed a normal kidney pelvis, calyces and ureter on the left side, and a large irregular filling defect in the upper third of the right ureter with a kink at the right ureteropelvic junction, pyelectasis and caliectasis. (Fig. 4.) A diagnosis was made of papillary carcinoma of the right ureter with possible involvement of the pelvis. On August 6th a right nephroureterectomy (complete) without partial cystectomy was performed. The patient made an uneventful recovery and was discharged on August 13th. The pathologic report was: (1) papillary carcinoma (transitional cell) of upper right ureter, low grade malignancy, (2) hydronephrosis, right, and (3) acute and chronic pyelonephritis.

The patient was again admitted to the hospital on November 4, 1953, with a small mass adjacent to the right nipple. He under-

went excision of a benign cyst of the right breast on November 10th and transurethral resection of the prostate on November 16th. He made an uneventful recovery and was discharged on November 24th.

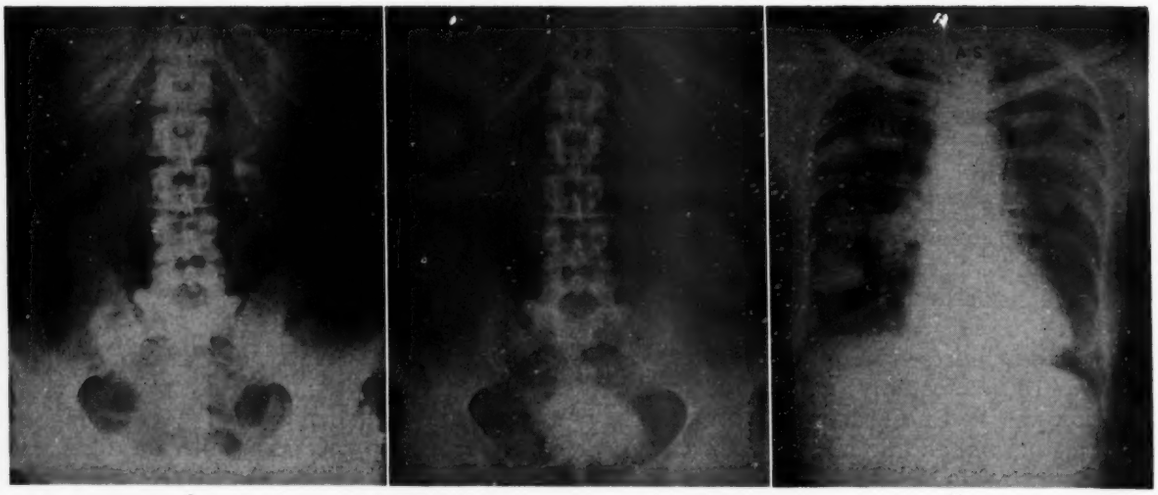
His third admission was on March 8, 1954, with a history of bleeding hemorrhoids for the past three months. A hemorrhoidectomy and excision of fistula-in-ano were performed on March 11th and he was discharged on March 18th. On April 16th and July 1st (eleven and fourteen months, respectively, after nephroureterectomy) he was in excellent condition and had no urinary symptoms. The urine was normal and no abdominal masses or tenderness were found.

Comment. It is interesting to note that an enlarged right kidney outline was observed in the roentgenograms taken during this patient's confinement one and a half years previously at another hospital. It is regrettable that no further investigation was made at that time, for in all probability the enlarged kidney represented hydronephrosis due to the unrecognized ureteral tumor. The urologist decided to perform complete nephroureterectomy without removing a cuff of the bladder because the primary tumor was situated in the upper end of the right ureter. An excellent result was obtained, for the patient was in perfect health fourteen months after operation with no evidence of recurrence of a tumor in the bladder.

Case IX. S. E. (Hosp. No. 108347), a sixty-one year old man, was admitted to the Sinai Hospital on September 24, 1953, with the complaint of hematuria, dysuria and diurnal frequency of ten days' duration. He had been treated for syphilis forty years before with salvarsan,® and had been in good health until the present illness. On examination the right pupil failed to react to light. The ankle jerks were absent bilaterally. Examination revealed chronic pulmonary emphysema, enlarged liver, benign hypertrophy of the prostate and generalized abdominal tenderness. The blood serologic tests for syphilis were negative. The urine showed albumin 1 plus, many red blood cells and an occasional pus cell. Urine culture was positive for Bacillus proteus. On excretory urography the plain plate showed an enlarged right kidney; the left kidney pelvis, calyces and ureter were normal but no dye was excreted from the right kidney in thirty minutes. At cystoscopy on September 25th a small tumor mass was seen protruding from the right ureteral orifice for a distance of 1.5 cm. A No. 5 F. catheter was passed up the right ureter and met an obstruction 2 cm. up; this was overcome and the catheter entered the pelvis to drain off 30 cc. of chocolate-colored urine. A No. 5 F. catheter was passed up the left ureter with ease. Bilateral retrograde ureteropyelograms were made and revealed a normal left kidney pelvis, calyces and ureter, and an irregular crescentic filling defect in the lower third of the right ureter extending for a distance of 3 cm. (Fig. 5.) Ureterectasis, pyelectasis and caliectasis were present on the right side. A tentative diagnosis was made of neoplasm of the lower right ureter. On September 29th a right nephroureterectomy with partial cystectomy was performed. The patient made an uneventful recovery and was discharged on October 8th. The pathologic report was: (1) papillary carcinoma (transitional cell) of the right lower ureter (low grade malignancy), (2) hydronephrosis, and (3) chronic pyelonephritis. The patient returned for examination on July 1, 1954 (nine months postoperatively), and was in excellent health. The urine was normal and examination of the abdomen revealed no masses or tenderness. Cystoscopy was entirely negative.

Comment. The protrusion of a piece of tumor through the ureteral orifice and the characteristic filling defect in the ureteropyelogram led to an early diagnosis and prompt radical surgery (one-stage combined nephroureterectomy and partial nephrectomy), which should yield an excellent long-term result in this case.

Case X. A. S. (Hosp. No. 113089), a seventy-three year old woman, was admitted to the Sinai Hospital on March 17, 1954, with the complaint of intermittent episodes of gross hematuria of five months' duration. Three years before she had been troubled with urinary frequency, urgency and burning but no hematuria. A complete urologic study had shown mild right hydronephrosis with ptosis and granular cystitis and trigonitis. There had been no evidence of tumor of the bladder or ureter and no further urologic treatment had been prescribed. Three weeks prior to the present admission she complained of marked anorexia and loss of weight, strength



6 7 8

FIG. 6. Case x. Excretory urographic film taken after thirty minutes showing a normal left kidney pelvis and calyces and a kink at the left ureteropelvic junction. No dye appeared in the right kidney.

FIG. 7. Case x. Retrograde ureteropyelogram attempted with a Braasch bulb catheter within the right ureteral orifice and with the patient in Trendelenburg's position. No dye passed up the right ureter but a sufficient amount was injected to give a good cystogram which showed an irregular filling and splinting of the right lateral wall of the bladder.

FIG. 8. Case x. Chest film showing extensive metastases in both lungs, more marked on the right side.

and appetite, diurnal and nocturnal frequency, and intermittent hematuria with clots. On examination she appeared to be a thin, emaciated, anemic woman. An enlarged right kidney was palpable. On pelvic examination an indurated elongated sausage-shaped mass was palpable on the right lateral aspect of the bladder. The urine was negative for albumin and contained innumerable red blood cells and pus cells. At cystoscopy (March 18th) a blood clot containing grayish tumor tissue was seen protruding from the right ureteral orifice and a piece of this tumor was removed with a Lowsley grasping forceps. Attempts to pass Nos. 4, 5 and 6 F. catheters up the right ureter were unsuccessful. A No. 8 Braasch bulb catheter was passed into the right ureter for a distance of 1 cm.; and after the patient was placed in Trendelenburg's position, 30 cc. of 20 per cent diodrast® were injected into the right ureter. A right ureterogram failed to show a long irregular filling defect in the lower right ureter, as no dye passed beyond the obstruction in the lower ureter. The diagnosis was a right ureteral malignant tumor. On March 19th a chest film showed diffuse "snowball" metastatic deposits scattered throughout both lungs. Films of the long bones and pelvis showed an osteoblastic (metastatic) area in the right ilium. Excretory urography on the same day revealed a normal left kidney pelvis, calyces and ureter but no dye excreted into the right kidney in thirty minutes. Operative treatment was contraindicated because of the patient's poor condition and diffuse pulmonary and osseous metastatic lesions. She was given a transfusion of 500 cc. of whole blood and was discharged on March 21st. A biopsy specimen from tissue in the right ureteral orifice showed aggregates of red blood cells, fibrin, necrotic debris and neoplastic cells of the papillary carcinomatous (transitional cell) variety. Her course at home was progressively downhill and she died on April 17th of cardiac failure and pulmonary metastases. The final diagnosis was papillary carcinoma (transitional cell) of the lower right ureter with metastases to both lungs and ilium. (Figs. 6 to 8.)

Comment. An interesting feature in this case was the absence of any demonstrable pathology in the upper urinary tract in the urologic study performed three years prior to admission to the hospital. Undoubtedly the ureteral carcinoma was of the rapidly growing variety, as the chief complaints of intetmittent hematuria, weakness, anorexia, etc., dated back only three months. The

diagnosis was established by obtaining a biopsy specimen from the portion of the tumor protruding from the ureteral orifice and by palpating an elongated, firm, sausage-shaped mass on the right lateral aspect of the bladder. Efforts to obtain a satisfactory ureteropyelogram were unsuccessful despite the injection of the dye through a Braasch bulb catheter with the patient in the Trendelenburg position. Apparently the tumor was of the infiltrating type and completely occluded the lower portion of the ureter. The presence of diffuse pulmonary metastases and a metastatic lesion in the bony pelvis and her extremely debilitated condition militated against employing any type of operative or deep x-ray therapy.

Case xi. L. T. C. (Hosp. No. 116688), a sixty-seven year old white man, was admitted to the Sinai Hospital on July 26, 1954, complaining of left flank pain of three years' duration and hematuria of two months' duration. He had been in good health until January, 1950, when dull pain developed in the left flank; this pain radiated around the umbilical area and down to the left groin. A urologic study was performed in an out of town hospital by another urologist and a diagnosis of a non-radiable calculus in the left ureter was made. Treatment consisted of dietary regulation, acidification of the urine and forced fluids, but no antibiotics or cystoscopic dilatation of the ureter was prescribed. The patient had had similar episodes of left renal pain in 1951, 1952 and 1953. He had been treated for diabetes without insulin since the early part of 1950. In the mid-portion of 1953 symptoms of prostatism ensued and a diagnosis of carcinoma of the prostate was made. A radical perineal prostatectomy and bilateral orchidectomy were performed elsewhere. Following the operations the patient was placed on estrogenic hormone therapy which he discontinued against advice after three weeks. In May, 1954, he had hematuria, accompanied with small clots, which persisted for three weeks. About four weeks prior to admission to this hospital he had a recurrence of the dull left flank pain. He had no urinary symptoms or loss of weight. The examination was essentially negative except for an enlarged, slightly tender left kidney. The prostatic area showed no evidence of malignancy. The blood pressure was 140/70. Urine was negative except for sugar 3 plus. A hemogram was normal. Excretory urography was performed on July 26th; films taken eight and fifteen minutes after intravenous injection of 20 cc. of diodrast showed the right kidney pelvis and calyces to be well filled and normal in outline. No dye appeared in the left kidney pelvis in the films taken at eight, fifteen and thirty minutes. The left kidney outline appeared to be twice the size of the right kidney. Cystoscopy on July 27th revealed a small papillary tumor (0.5 cm. in diameter) on the left lateral wall of the bladder near the vesical orifice; this tumor was removed with a No. 24 McCarthy resectoscope. No blood was seen escaping from the left ureter, nor was any tumor visible at the left ureteral orifice. Nos. 5 and 6 F. ureteral catheters met an impassable obstruction at the left ureterovesical orifice. An attempt was made to obtain a ureteropyelogram using a No. 6 Braasch bulb catheter but no dye passed up the left ureter. Cystoscopic examination was repeated on July 30th; the same impassable obstruction was encountered and no dye passed up the ureter. A tentative diagnosis was made of a left ureteral obstruction with a non-functioning hydronephrosis, and the possibility of a primary ureteral neoplasm in the lower left ureter was suggested as the causative lesion. On August 3rd a combined one-stage nephroureterectomy and partial cystectomy was performed through a lumbar incision and subsequent left Gibson incision. The postoperative course was uneventful and the patient was discharged on August 13th. The pathologic report was: (1) primary papillary carcinoma (transitional cell) of lower left ureter, (2) hydronephrosis, (3) hydro-ureter and (4) papillary carcinoma (secondary) of the bladder.

Comment. It is too soon to predict the outcome in this case. It is regrettable that the correct diagnosis was not made at the time of the first cystoscopic study in 1950, for it is most likely that the lesion responsible for the left flank pain was unrecognized primary papillary carcinoma of the lower left ureter. Four years elapsed before the patient sought other urologic assistance and a secondary tumor of the bladder had developed and presented symptoms which led to further investigation and establishing of the correct diagnosis.

Case xii. C. R. (Hosp. No. U-120322), an eighty-one year old white man, was admitted

to the Sinai Hospital on December 6, 1954, complaining of gross hematuria of three months' duration. The hematuria was painless and intermittent, and was accompanied with a weight loss of 10 pounds. Examination was negative except for signs of generalized arteriosclerosis, arteriosclerotic heart disease and benign enlargement of the prostate. The urine was grossly bloody. At cystoscopy on December 7th the positive findings included trilobar hypertrophy of the prostate, trabeculated bladder and bloody urine escaping from the right ureteral orifice. A No. 6 F. catheter was passed up the left ureter to the pelvis with ease but a similar catheter met an obstruction 10 cm. up in the right ureter; this was overcome and clear urine was drained. Bilateral retrograde ureteropyelograms showed a normal left kidney pelvis, calyces and ureter; however, on the right side there was moderate hydronephrosis with a filling defect in the middle third of the right ureter and dilatation of the ureter above the defect. A diagnosis of primary neoplasm of the right ureter was made. On December 16th a right nephroureterectomy (complete) was performed but no cuff of the bladder surrounding the ureter was removed. The postoperative course was uneventful and the patient was discharged on December 30th. The pathologic report was: (1) papillary transitional cell carcinoma of the right ureter, (2) right hydronephrosis, (3) right pyelonephritis, chronic, and (4) arteriosclerotic scarring, right.

Comment. The diagnosis was readily established in this case. Complete nephroureterectomy was performed without partial cystectomy because of the advanced age of the patient. One month following operation he appeared to be in excellent condition, with no urinary symptoms or evidence of recurrence.

Benign Ureteral Tumor

Case XIII. R. R. (Hosp. No. 14109), a forty-three year old white man, was admitted to the Sinai Hospital on May 12, 1940, with the complaint of left renal colic of one day's duration. He had had a sudden sharp pain in the left loin which radiated to the left lower quadrant and which was recurrent. This required injection of morphine (¼ gr.) and atropine (1/150 gr.) for relief. The patient had been troubled with arthritis of the shoulders for the past three years. Examination was negative except for tenderness in the left lower quadrant and left loin. Examination of the urine was negative except for many red blood cells (microscopic). At cystoscopy on the day of admission the bladder was normal. A No. 6 F. catheter was passed up each ureter with ease, but on the left it met an obstruction at about 20 cm. Blood-tinged urine was obtained from the left catheter. A plain film (K. U. B.) showed a calcified oval shadow (1 cm. diameter) in line with the left ureter, opposite the transverse process of the second lumbar vertebra. Plain films (stereoscopic) with catheters in the ureters showed the tip of the left catheter to be in contact with the calcified shadow at the ureteropelvic junction. A left pneumoureteropyelogram showed the air around the shadow. On bilateral pyelograms a normal right kidney and ureter were seen and the calcified shadow was located at the ureteropelvic junction. The diagnosis was a stone in the left ureter (at the ureteropelvic junction) and the patient was advised to have operative removal of the stone. On May 14th a renal exploratory operation was performed through a lumbar incision. A pyelotomy incision was made but no stone was found. Several small cortical incisions were made in order to explore each calyx but no stone was found. On the superior and medial aspect of the ureter at the ureteropelvic junction there was a non-infiltrating small papillary growth (about 1 cm. in diameter) covered with blood and calcareous material. In view of the malignant potentialities of this tumor it was decided to perform a nephrectomy and partial ureterectomy. The convalescence was uneventful and the patient was discharged on May 27th. The pathologic report was: (1) benign papilloma of the upper ureter with calcification, and (2) chronic recurrent pyelonephritis.

On September 18, 1941, he had severe right renal colic accompanied with fever, chills and pyuria. He was rehospitalized on September 21st. Urologic study revealed a calculus in the upper third of the right ureter, with moderate pyelectasia and caliectasis. A right ureterolithotomy was performed on September 23rd, and after an uneventful convalescence the patient was discharged on October 10th. For the next six years he returned at intervals of six months and remained in good health. Subsequently, he has reappeared yearly for examination, the last visit being on May 4, 1954 (fourteen years postoperatively), when he was found to be in

excellent health with no complaints. Examination of the urine was negative.

Comment. This case was the only instance of benign ureteral tumor in more than 10,000 urologic admissions to this hospital. The tumor, which was a calcified papilloma, was incorrectly diagnosed as a stone in the upper third of the ureter. At operation a careful search of the ureter, pelvis and calyces was made but no stone was found. A small pea-sized papilloma covered with blood and calcium salts was found in the upper portion of the ureter close to the ureteropelvic junction. Although the tumor was of the non-infiltrating type, nephrectomy and partial ureterectomy were performed because of the malignant potentialities of benign papilloma. Sixteen months later a large stone was removed from the upper third of the right ureter by ureterolithotomy. This patient has been followed up for fourteen years and there has been no recurrence of stone or tumor.

CONCLUSIONS

A review of 454 primary malignant tumors of the ureter and 138 benign tumors, which includes the one benign and twelve malignant cases presented herein, warrants the following conclusions:

1. Primary tumors of the ureter are uncommon but by no means rare clinical and pathologic entities.

2. The malignant type of tumor is more common than the benign (3:1). The incidence of ureteral tumors in urologic practice is 1:3,690 cases, and in autopsy series 1:10,000.

3. The malignant ureteral tumor occurs most frequently in the sixth, seventh and eighth decades of life, with the highest incidence in the seventh decade. The benign tumors are seen earlier, the majority occurring in the fourth and fifth decades.

4. The malignant tumor occurs more frequently in men than in women (2:1), whereas the benign tumor shows only a slight preponderance in men.

5. The malignant tumor shows no special predilection for either ureter but the benign type appears to involve the left ureter more frequently.

6. The lower third of the ureter is the most common site for both types of tumors. The incidence decreases as the ureter is ascended.

7. Ureteral tumors are more often single than multiple, (i.e., malignant 6:1, benign 4:1) and are rarely bilateral.

8. A wide variety of pathologic types of malignant and benign tumors has been described. The most common form of malignant tumor is transitional cell papillary carcinoma (72 per cent) and next is squamous cell carcinoma (7 per cent). In the benign group the order of frequency is papilloma (50 per cent), fibroma or polyp (28 per cent) and vascular tumors (10 per cent).

9. The etiology of ureteral tumors has not been clearly established. One or more of the following factors may be responsible: obstruction, trauma or irritation, infection, hyperplastic epithelial changes, specific carcinogens of exogenous or endogenous origin, and hormonal imbalance. Our knowledge of the pathogenesis of primary ureteral neoplasms has been considerably enhanced by the experimental work of Scott and Byrd who, for the first time, were able to produce primary ureteral carcinoma following the administration of beta naphthylamine.

10. Several theories have been offered to explain the spread, multiplication and recurrence of ureteral neoplasms. The most logical explanation appears to be the theory of the multicentric origin of urothelial tumor which was originally proposed by Ewing and which implies that several centers in different parts of the urothelial tract may be affected by specific though unknown carcinogens and produce neoplastic cells that appear simultaneously or at different intervals of time and that may coalesce or remain as discrete tumors.

11. There is no symptom complex characteristic of ureteral tumors, and both types may present the same clinical picture. The outstanding symptoms are hematuria (75 per cent), pain (36 per cent) and tumor mass (10 per cent). Since this triad of symptoms is infrequently present in the same case, each symptom *per se* bears no great diagnostic significance.

12. The length of time elapsing between the onset of symptoms and the establishment of the correct diagnosis was eight months in the benign group and 24.2 months in the malignant group.

13. The most important diagnostic aids are (*a*) staining of urinary sediment for tumor cells, (*b*) cystoscopy and (*c*) retrograde ureteropyelography.

14. The only absolutely positive evidence at cystoscopy is the presence of a tumor protruding through the ureteral orifice. Histologic study of a piece of the tumor obtained by any biopsy method is invaluable in establishing the correct diagnosis.

15. The following cystoscopic findings may be regarded as presumptive evidence of a ureteral tumor: (*a*) blood escaping from the ureteral orifice, (*b*) intraureteral obstruction to the passage of a catheter, (*c*) increased ureteral bleeding following passage of a ureteral catheter (Chevassu-Mock's sign), (*d*) drainage of clear urine when the catheter passes beyond the obstruction (Marion's sign), (*e*) presence of a small papillary tumor in the bladder near the ureteral orifice, (*f*) other bladder signs such as elevation of the intramural portion of the ureter, gaping orifice and edema or congestion about the orifice.

16. Retrograde ureteropyelography is the most dependable diagnostic aid. The characteristic signs of a ureteral tumor are narrowing or obliteration of the outline of the ureter at the point of obstruction, and an accompanying filling defect. Solid tumors (i.e., infiltrating papillary carcinoma or squamous cell carcinoma) usually produce an irregular diminution in the caliber of the ureteral lumen at the site of the tumor, with ureterectasis, pyelectasis and caliectasis above. The solitary pedunculated papilloma or polyp produces a ring-shaped or goblet-shaped defect in the outline of the ureter accompanied with dilatation of the ureter above the tumor area and occasionally dilatation of the ureter below the tumor area.

17. Benign and malignant tumors of the ureter may easily be mistaken for other lesions and should be differentiated from the following conditions: (*a*) non-opaque ureteral calculus, (*b*) blood clot in the ureter, (*c*) papillary carcinoma of the renal pelvis with a secondary implant or metastases in the ureter and (*d*) obstruction of the ureter by an inflammatory process or a neoplastic mass of intrinsic or extrinsic origin.

18. The operative treatment of ureteral tumors poses a serious problem as evidenced by the numerous operations employed and the relatively poor results obtained. The choice of operation may be considerably influenced by one or more of the following factors: general condition of the patient, functional status of the opposite kidney, type and site of the tumor, and technical difficulties encountered at operation.

19. In the case of a malignant tumor the operation of choice is a one-stage complete nephroureterectomy combined with partial cystectomy (i.e., removal of a cuff of the bladder surrounding the ureteral orifice). The same operative treatment is also indicated in cases of so-called benign papilloma, notwithstanding the good results obtained by conservative operations as reported by several urologists.

20. Other benign tumors of the ureter such as polyp, fibroma and vascular tumors, whose histologic diagnosis has been definitely established by biopsy at cystoscopy or operation, may be treated by conservative surgery: (*a*) transvesical or transureteral (ureterotomy) fulguration, excision or resection, or (*b*) segmental resection with end-to-end anastomosis or reimplantation of resected ureter into the bladder, bowel or skin.

21. The best results in either type of tumor are directly dependent upon early diagnosis and prompt surgical treatment. In the past ten years there has been a remarkable improvement in the operative treatment of ureteral tumors from the standpoint of operative mortality and morbidity. There were no operative deaths in sixty-one cases of benign tumors and only nine deaths (4.7 per cent) in 190 operations for malignant tumors.

22. The results of the operative treatment of malignant tumors are rather disappointing from the standpoint of curability and longevity despite the fact that accurate and careful follow-up studies were available in only 109 operative cases and that in many of these cases the patients were not followed up for sufficiently long periods to warrant definite conclusions. In the group of seventy-one patients reported living after operation only ten patients survived more than five years. In the group of thirty-eight patients who survived operation but eventually succumbed, thirty-five were dead before three years had elapsed.

23. The best results in the malignant group from a curative and longevity standpoint were obtained in those patients treated with one-stage nephroureterectomy and partial nephrectomy. The incidence of recurrence of tumor

in the ureter and bladder was also lowest in this same group.

24. In view of the malignant potentialities of benign urothelial tumors and the tendency of malignant urothelial tumors to recur or multiply, every patient with a benign or malignant papillary tumor of the ureter (or of the bladder or renal pelvis) should be impressed with the absolute necessity of returning for periodic re-examination (including cystoscopy) for the remainder of his life, i.e., at least every three months for the first two years and every six months thereafter.

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Oncocytic Cell Adenoma of the Parotid Gland

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ONCOCYTIC cell adenomas of the salivary glands are rare but distinct histologic tumors. A review of the literature reveals sixteen well documented cases. Much confusion still exists regarding the nature of salivary gland tumors, but as the oncocytic cell adenoma is quite distinct, it should be readily recognized. Believing that greater familiarity with the characteristics of this tumor will result in an increased incidence of its recognition, we are reporting a case and briefly reviewing the literature.

CASE REPORT

A fifty-six year old woman was admitted to the Jackson Clinic in August, 1953, because of a small tumor of the left parotid gland. The lesion was visible as a firm, spheric mass, approximately 2 cm. in diameter, under the angle of the jaw. It was firm and non-tender and could barely be moved over the subjacent tissue to which it seemed to be attached. There was no evidence of facial nerve involvement and no lymph nodes were palpably enlarged. Routine physical examination showed mild hypertension, without signs or symptoms of cardiac decompensation. A clinical diagnosis was made of a mixed tumor of the left parotid gland.

On August 10, 1953, the tumor was removed under local anesthesia. A transverse incision under the angle of the jaw disclosed a well encapsulated, lobulated tumor, deeply embedded in the parotid gland. It was possible by careful excision to remove the entire tumor without damage to the seventh nerve. The gross specimen was a bosselated, firm, ovoid mass measuring 3.0 by 2.5 by 1.5 cm. surrounded by a thin capsule which had been previously torn at one point. The cut surfaces were white, tinged with pink, lobulated and slightly bulging. Microscopic examination revealed a solid tumor composed of uniform cells arranged in cords and columns separated by narrow septa forming an alveolated pattern. The individual cells were large and round or polygonal with eosinophilic, finely granular cytoplasm. Their nuclei were small, round or oval and contained fine chromatin material. (Fig. 1.) Fragments of salivary gland accompanied by a scanty lymphoid infiltrate were present in the outer portion of the capsule, and several cross sections of a normal duct were noted in the connective tissue septa dividing the tumor into lobules. No mitotic figures were found in the sections examined, and there was no evidence of capsular or venous invasion. The cells of the tumor fit the description of oncocytes (or onkocytes) given by Schaffer[1] and Hamperl.[2,3]

Deep radiotherapy was ordered after a preliminary report of adenocarcinoma of the parotid gland was made, and the patient received 3,000 r in a sequence of ten treatments. The diagnosis was revised to oncocytoma, benign, when another pathologist (E. L. S.) reviewed the sections and recalled having seen a section of a similar tumor in a seminar discussed by Willis in 1949.†

† This was the case of a fifty-eight year old man submitted by Dr. Robert S. Haukohl, Evangelical Deaconess Hospital, Milwaukee, Wisconsin, to the Tumor Seminar of Wisconsin Society of Pathologists, December 10, 1949, Milwaukee, with Rupert A. Willis as moderator.

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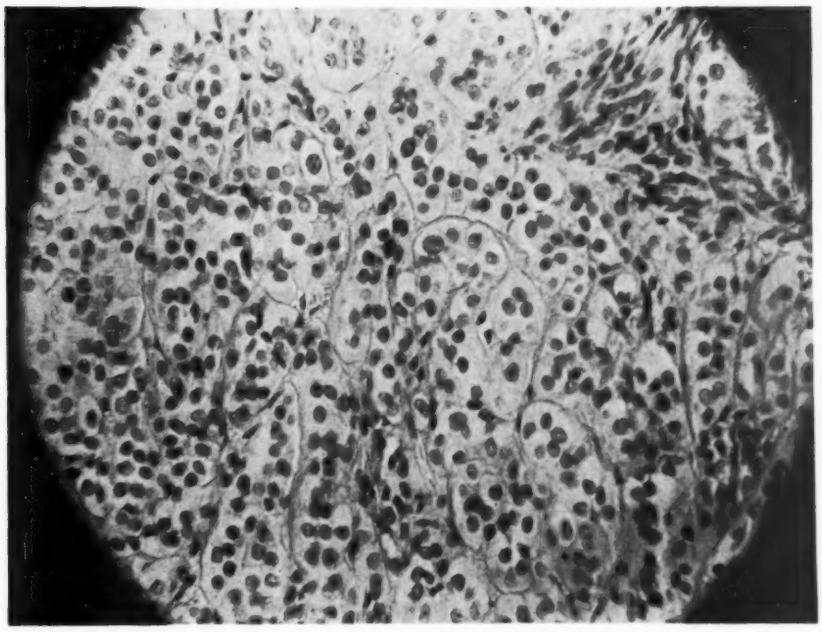

FIG. 1. Photomicrograph of oncocytic adenoma showing characteristic cells forming a solid tumor and presenting an alveolated pattern (original magnification × 350).

THE NATURE OF ONCOCYTIC CELLS

The characteristic large, eosinophilic cell known as the oncocyte, or onkocyte, was first described by Schaffer,[1] but Hamperl[2,3] made the first complete study of them and originated the term of onkocyte from a Greek word meaning "I become bigger." Since oncocytes are not peculiar to the parotid gland but can be found in various epithelia, they represent a cytologic entity. Their general characteristics are given in the following résumé: They appear as large, usually well outlined cells, spherical or polyhedral, depending on the pressure exerted upon each other. Hematoxylin-eosin stain produces a bright acidophilic coloration of the cytoplasm which is finely granular. The nucleus is small, regular in shape, round and vesicular, or at times the nucleus may be pyknotic, hence the name "pyknocyte" given to the cell by Zimmerman.[4] Usually the cells have a single nucleus, and on rare occasions they may contain two nuclei or a bilobed nucleus.[5] Mitotic figures are unusual, but have been observed in some cases.[6] Hamperl[2,3] believes that they propagate by amitotic division.

According to Hamperl,[6] additional information about the cells may be gained from special stains. Masson's trichrome stain shows greenish red granules in the cytoplasm; in some cells these are interspersed with golden yellow granules which may represent degenerative changes.[7] Mallory's aniline blue stains the granules purplish blue. Feyrter[8] believes the "inclusion stain" for metachromasia is specific. With this method, a few drops of 1 per cent thionine or cresyl violet solution in 0.5 per cent aqueous tartaric acid is placed on a thin section, obtained by frozen section, and a cover glass is added and sealed. The oncocytes' property of staining with the inclusion stain is apparently due to certain lipids which they contain.[6] Since they are metachromatic, the oncocytes are easily identified by this method. Meza-Chávez[5] was unable to demonstrate glycogen or mucin in these cells.

Oncocytes may be interspersed between normal parenchymal cells in normal glands. They have been found in the thyroid and parathyroid glands, the salivary glands, the anterior and posterior lobe and the stalk of the hypophysis, the pancreas, the liver, the esophagus and stomach, the pharynx, trachea,[2,3] and also in the bronchus,[9] the uterine tubes and testis,[2] and in the wall of the lacrimal sac.[10,11]

It is noteworthy that oncocytes are found in individuals of the older age group. That observation originally made by Hamperl[3] was

confirmed by Meza-Chávez,[5] who studied 100 normal parotid glands and found oncocytes only in subjects over forty-nine years of age. Oxyphilic cells have been found in the parathyroid of a seven year old patient and they appear quite regularly in these glands in late childhood, but this seems to be the exception.[12] Among the reported cases there are oncocytic adenomas of the pancreas,[13] several of the anterior lobe, posterior lobe and stalk of the pituitary,[3] the bronchus,[9] the adrenal and renal cortex,[14] and the wall of the lacrimal sac.[10,11] Hamperl suggested that the Hürthle cell tumors of the thyroid and the eosinophilic tumors of the parathyroids should be regarded as oncocytic cell adenomas. Oncocytic cells have been found as the epithelial component of the papillary cystadenoma lymphomatosum.[15] Hamperl[3] found a cyst of the larynx lined with oncocytic cells.

Various names have been assigned to the acidophilic cells of different organs by authors who did not recognize the existence of comparable cells in other epithelia. Thus, those of the parathyroids are known as oxyphilic cells of Welsh, those of the thyroid have been called Hürthle cells by some authors and Askanazy cells by others, and those in the salivary glands have been referred to as acidophilic, eosinophilic or oxyphilic cells as well as oncocytes. Ewing[16] referred to oncocytes as "Hürthle cells," a name which prevails in American literature, but Hamperl,[6] an authority in this field, has claimed that this concept is incorrect. "Hürthle cells" were described by Nonidez[17] as the interfollicular (parafollicular) cells of the dog. They have their own characteristics (different from oncocytes) and a neoplasm arising from the interfollicular cells has never been proved, contrary to a report by Bakay[18] in 1948. Similarly, the origin of the so-called Hürthle cells has been misunderstood by authors who have considered them separately in each organ and not as similar cells regardless of origin. Eisenberg and Wallerstein[19] and Getzowa[20] thought that "Hürthle cells" arose from the parathyroid because of their resemblance to the oxyphilic cells of that organ.

Hamperl's hypothesis[6] seems far more logical. He considered that "the onkocytes are always the products of a transformation of the parenchymal cells of their respective organs." He noted a transition from thyroid adenomas, containing scattered oncocytes, to adenomas composed entirely of them. He claimed that this transformation can also be seen in carcinomas. Early in 1935 Clute and Warren[21] said: "The similarity between these cells (oncocytes) and certain of the changes in thyroid cells most frequently encountered in involution following primary hyperplasia, led us to follow Ewing and the others in considering this tumor [a so-called Hürthle cell carcinoma] as a true thyroid carcinoma rather than as of parathyroid origin, as suggested by Eisenberg and others. While admittedly the cells resemble the oxyphil cells of the parathyroid, they appear to resemble even more closely the acidophilic phase occasionally encountered in the thyroid epithelium itself."

Wilensky and Kaufman[22] regarded Hürthle cells as representing a conversion of the histologic appearance from that of the more usual type of adenocarcinoma to the Hürthle cell type. Harris[23] pointed out the transition from normal cells of a duct to oncocytic cells in an oncocytic cell adenoma of the parotid gland. Recently Meza-Chávez[5] called attention to various stages of transformation from normal parotid epithelium into oxyphilic granular cells in his study of oxyphilic cell adenomas of the parotid gland. This article is well documented with excellent photomicrographs. Some authors[23–25] claim that oncocytic cells are ductal in origin, but the previously mentioned photomicrographs clearly show that acinous cells may undergo oncocytic transformation.

The significance of these changes in morphology is not well understood. To Hamperl[6] oncocytes represented a "peculiar degeneration." Others[5,7] have regarded these changes as a stage in further differentiation or in an aging process. Meza-Chávez[5] reported granular eosinophilic material in the duct of an oxyphilic cell adenoma of the parotid gland and concluded that oncocytes are "able to secrete, or at least to extrude, part of their cytoplasmic content." Jaffé thought of the change as "a progressive cellular differentiation peculiar to senescence." The generally accepted hypothesis is that the advancing age of the cell produces a change in the staining reaction and size of the cell.[5,26]

REPORTED CASES OF ONCOCYTIC CELL ADENOMA OF THE SALIVARY GLAND

Only a few well documented cases of oncocytic cell adenoma of the salivary glands have

been reported. We have been able to find sixteen acceptable cases in the literature. Each tumor was considered benign, but recently Buxton, Maxwell and French[27] have described an acidophilic-celled carcinoma of the parotid gland.

Ackerman,[7] in a review of the literature in 1943, found seven cases which he regarded as being oncocytomas (oncocytic cell adenomas) and he added one, making a total of eight cases. The cases that he accepted as being well documented and characteristic of oncocytoma were those of Gruenfeld and Jorstad,[24] McFarland,[28] Steinhardt,[29] Ahlbom,[30] Škorpil's Case IV,[25] and Stöhr and Risak (two cases).[31] The latter authors called one of their two cases a "tubular adenoma." All tumors occurred in individuals of an advanced age; the youngest was fifty-six, the oldest eighty-five. There were no follow-up studies in two cases, the tumor recurred in two instances, and in none was the tumor considered malignant.

A few years after Ackerman's report, Stump[12] made a detailed study of oncocytic adenomas, and after a critical review of the literature he deleted the two cases of Stöhr and Risak and added four cases[32–35] and one of his own, making a total of eleven acceptable cases. Hückel[33] had reported three cases, only one of which (Case 1) was accepted by Stump; McFarland's second case[35] was considered as one "in view of the wide experience of this author" in spite of the fact that it was reported without microscopic description. One of Blair and Olch's two cases[34] was accepted because it had been examined by Gruenfeld and Jorstad and found identical to their case. Case VIII of Lloyd's paper[32] on salivary adenoma and adenolymphoma was an oncocytic adenoma of the parotid gland.

In 1949 Meza-Chávez[5] reported five additional cases of oxyphilic cell adenoma. (He used the term, oxyphilic granular cell, in preference to oncocyte.) His work referred to some of the earlier cases[8,23–25,28,30,32] which he considered as belonging to this group. He regarded Hückel's Case 1,[33] one case reported by Blair and Olch,[34] McFarland's second case,[35] and a case reported by Duplay[36] as probable examples of oxyphilic adenomas, but he rejected the two reported by Stöhr and Risak (as did Stump), one reported by Steinhardt and one by Franssen.[37] A case reported by Harris[23] in 1936 was included in Meza-Chávez' series but was absent from the reports of Ackerman[7] and Stump.[12]

Christopherson[38] reported a case of oncocytoma in 1949, but he described small cartilaginous foci around the periphery of the tumor. Such mesenchymal elements are thought to be unusual in true oncocytic adenomas, and there is a question in the authors' minds if this were not a mixed tumor in which the epithelial component had undergone oncocytic transformation.

Reference to six additional cases regarded as oncocytic cell adenomas of the parotid was found, but none of these was described in detail. In 1951 Kirklin, MacDonald, Harrington and New[39] reported three such cases in a review of 717 cases of parotid tumors. Two years later Buxton, Maxwell and French[27] included three cases in their series of epithelial tumors of the parotid gland. The latter paper is accompanied by an excellent colored photomicrograph which shows the main features of the "acidophilic-celled" adenoma. Since the cases published by Meza-Chávez were from the files of the University of Michigan, it seems possible that the three cases referred to in the article by Buxton et al.[27] of the same institution may have been included in the original article. The article of Buxton and his group also referred to nine cases of "acidophilic gland-celled" carcinomas but these are not described.

CLINICOPATHOLOGIC FEATURES

A summary of the reported cases indicates that oncocytic cell adenomas occur in both sexes with equal frequency. All cases in which age is specified belong to the older age group, with the exception of Meza-Chávez' fifth case in which the patient was thirty-two years old. These tumors are encountered infrequently; Kirklin et al,[39] found three among 717 parotid tumors. Buxton and his group[27] found three cases in a series of 227 epithelial neoplasms of this gland. Rawson et al.,[40] in a review of 160 tumors of the salivary glands, found no oncocytic cell adenomas, but mention was made of foci of oncocytic cell transformation in mixed tumors of the parotid gland. One of us (A. S. J.), who has had thirty-five years' experience in dealing with cases confined chiefly to tumors and cysts of the neck and face, has never before seen this particular tumor. All reported cases except one, possibly two (if Duplay's case is accepted), have been

located in the parotid gland. The exception was an oncocytic cell adenoma arising in the salivary gland tissue of the hard palate.[30] The tumor reported by Duplay was located in the submaxillary gland.[36]

The clinical appearance of these tumors is that of a benign tumor indistinguishable from the common mixed tumors. They are firm, small nodules usually under 3 to 4 cm. in maximum diameter. Attachment to the skin, pain and facial palsy have not been reported. Exploration of the regional lymph nodes has not yielded evidence of metastases. Recurrences, when excised and examined, were exact replicas of the original growth. In some instances the tumor had been present for more than ten years before removal and had remained without change during that time. Until recently it was thought that oncocytic cell transformation was a sign of benignancy, but Buxton and his associates have shown that such an affirmation can be highly criticized. Although they offered no proof that the "acidophilic-celled carcinoma" arises from the corresponding adenoma, they believed that the three cases of many years' standing favor this possibility.

Oncocytic cell adenomas do not have sufficient semeiologic characteristics to distinguish them from simple mixed tumors. The recognition of these tumors rests with the pathologist. Macroscopic examination reveals an encapsulated, solid, white or pale pinkish gray tumor. Cut surfaces may be faintly lobulated and bulging. The typical eosinophilic cells, on histologic examination, are arranged in cords or alveolated masses, or in a tubular or acinous pattern. Both arrangements may be present in the same tumor. Ducts are infrequent, often dilated and sometimes contain a granular eosinophilic substance. Arterioles and capillaries are present, but the extravagant circulation of a cancerous tumor is lacking. Lymphocytic infiltration and hemorrhagic and degenerative areas have been described. Noteworthy are the foci of oncocytic cell hyperplasia which may be seen in the normal tissue surrounding the adenoma. These are of practical importance since it is from these foci that new adenomas may arise after removal of the main tumor and account for the so-called recurrences which have been reported.

Brief attention should be called to several other types of salivary gland adenomas to avoid confusion. Godwin and Colvin[41] apparently described a case of serous cell adenoma which they stated was different from oncocytic cell adenoma, but they failed to identify it further. Buxton and his group[27] gave a clear description of these adenomas as differentiated from the "acidophilic-celled" adenoma. "Unlike the acidophilic-celled adenoma, the cytoplasm of these cells has an affinity for the basic dye, hematoxylin. Small amounts of mucin are present in dilated spaces . . . " Another tumor which must not be confused with the adenoma is that enigmatic tumor, papillary cystadenoma lymphatosum (Warthin's tumor). Although the epithelial component of these tumors may at times be formed by eosinophilic cells, they represent a distinct entity and should be classified separately.

The treatment of oncocytic cell adenomas is surgical excision. McFarland[28] applied radium but it did not eradicate the tumor. Most authors advise adequate local excision. Following removal, recurrences have been observed in some instances. McFarland's first case recurred after twelve years. Many regard these recurrences as arising from additional foci of oncocytic cells. If cases of recurrence prove numerous, wide excision of the gland might be the best treatment. Postsurgical irradiation has been administered twice (Harris' and authors' cases); in both instances no recurrence has been noticed, but these observations are too limited and the time in the latter case too short (two years) to warrant any conclusions about this mode of therapy. The problem of possible malignant degeneration arises from Buxton's observations. Although such a possibility has not been proved, it must never be overlooked and the tumor should be entirely excised.

TERMINOLOGY

We hesitate to enter the controversy regarding the name that should be applied to these tumors, but we wish to defend our position in referring to them as oncocytic cell adenomas. We have no special quarrel with the term, "oxyphilic granular cell adenomas" used by Meza-Chávez, but it has no distinct advantage over other descriptive terms such as acidophilic or eosinophilic granular-celled adenoma, which have also been applied to these tumors. The term, pyknocytoma, suggested by Harris does not seem appropriate for the obvious reason

that the cells are not pyknotic. The aforementioned terms, with the exception of pyknocytoma, were proposed after the term oncocytoma or oncocytic cell adenoma had already been used by Gruenfeld and Jorstad,[24] Škorpil[25] and Ackerman.[7] These descriptive terms merely add to the profusion of names being applied to this tumor. Meza-Chávez argued that the Greek word from which oncology and other words pertaining to tumor are derived gives an entirely different connotation to the word oncocyte than the impression of large size which Hamperl[2,3] tried to convey with this word. As a matter of fact, the Greek word from which Hamperl derived the term merely meant large size or bulkiness. With this interpretation in mind, the word oncocyte (or onkocyte) should be acceptable, and a tumor made up of these cells would logically be an oncocytic cell adenoma.

Stump[12] suggests applying the term "onkocytic adenoma" to tumors made up of "onkocytes" regardless of site, such as, "onkocytic adenoma of parotid gland," "onkocytic adenoma of thyroid." Others object to the term "oncocytoma" or "onkocytoma" on the grounds that it was first applied to an entirely different tumor by Jaffé[15] in 1932, and according to priority this name should be reserved for the adenolymphoma (Warthin's tumor) which has undergone oncocytic transformation. In this respect, confusion does exist regarding the term oncocytoma, or oncocytic cell adenoma. A request for literature and references on oncocytic cell adenoma from a large medical library brought twelve to fifteen articles, at least three-quarters of which were on adenolymphomas or papillary cystadenoma lymphomatosum without any reference to oncocytes or oncocytic cells. Most investigators do not recognize the terms as being synonymous, and Jaffé himself interpreted his case as aberrant salivary gland within a lymph node, stating, "The lymphatic tissue is not an essential part of the tumor, but apparently the remnant of a lymph node." If this view is accepted, then the term "oncocytoma" or "oncocytic cell adenoma" is applicable to any tumor composed of oncocytes.

SUMMARY

Because of the rarity of salivary gland oncocytic cell adenomas and the danger that this tumor may be mistaken for other entities of the salivary gland of a more serious import, we have reported this single case and briefly reviewed the salient features as presented in the literature.

The oncocytic adenoma reported in this article brings the total of acceptable authentic cases to seventeen. It is possible that additional cases are masquerading under other names and have been overlooked, and that even a larger number have not been reported.

We have presented arguments favoring the use of the term "oncocytic cell adenoma" for tumors composed of oncocytic cells.

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Malignant Mixed Tumor of the Parotid Gland with a Rare Metastasis

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One does not think of mixed tumors of the parotid gland as tumors that are prone to metastasize; this type of tumor usually becomes a surgical problem because of repeated local recurrences. Nevertheless, several cases have been reported wherein salivary gland tumors have metastasized widely.

Mulligan[1] reviewed the literature and found reports of metastases to the pleura, petrous portion of the temporal bone, lungs, cervical lymph nodes, liver, kidneys, pelvic peritoneum, ovaries, dura mater, bronchial lymph nodes, manubrium sterni, right seventh rib, right hip joint, femur, pubis, tracheal lymph nodes, retroperitoneal lymph nodes, clavicle, adrenals, leptomeninges, spleen, left diaphragmatic leaf, skull bones, left temporal lobe of the cerebrum, gallbladder, subcutaneous tissue, omentum and retroperitoneal tissue. He presented a case of his own in which a cancerous mixed tumor of the parotid metastasized to the pleura, lungs, liver, kidneys, ribs and lumbar vertebrae. The author also analyzed twenty other cases of his own in which metastases were present in the lungs, pleura, liver, bones, lymph nodes and spleen.

Perrin[2] presented a review of the literature as well as a case of his own. He observed a thirty-five year old white man who had a mixed tumor of the right parotid gland with eventual metastases to the lungs, liver and retroperitoneal tissue.

Macintyre[3] recorded a case of salivary gland mucoepidermoid carcinoma in a forty-six year old Negro woman. At autopsy metastases were found in all ribs, in the perilaryngeal lymph nodes, pericardium, myocardium, omentum, mesentery, liver and left adrenal gland.

In view of these facts concerning observed widespread metastases of malignant salivary gland tumors, we believe that the following case is worth reporting for three reasons: First, the tumor was unusually large and there was a history of duration of between twenty-five and thirty years. Second, histologic examination demonstrated a marked variation in pattern, from the relatively benign so-called mixed tumor of the parotid to a picture of complete malignant anaplasia. Third, the tumor metastasized to the lungs and to the subcutaneous tissues of the tip of the left middle finger; the latter metastasis is rare in any type of malignant tumor.

CASE REPORT

E. K., a sixty-eight year old woman, entered the Roger Williams General Hospital on May 17, 1953, because of a huge pendulous mass on the right side of her face. This mass had been present for twenty-five to thirty years and had been painless until the time of admission. However, throughout the years there had been a slow increase in size associated with slowly progressive facial paralysis. (Fig. 1.)

Her family and past personal histories were irrelevant. About twenty-five to thirty years before she had noticed a slight swelling just anterior to and beneath her right ear. At this time surgical removal had been advised but the patient had refused. After many years of slow growth of the tumor, in view of increasing pain, skin necrosis and progressive facial paralysis, the patient decided to permit removal of the tumor.

Physical examination showed a well developed, well nourished elderly white woman resting easily in bed. A huge mass protruded from the right side of her face, head and neck. There was evidence of right-sided facial paralysis. Her lungs and heart were normal on physical examination, as were the other organs and systems.

Laboratory studies revealed 5.22 million erythrocytes per cubic millimeter, 6.2 thousand

FIG. 1. Patient with malignant mixed tumor of the parotid gland of twenty-five to thirty years' duration.

leukocytes per cubic millimeter and 14.7 gm. per cent of hemoglobin. The blood sugar was 126 mg. per cent and the blood non-protein nitrogen was 40.5 mg. per cent. The thymol turbidity test showed less than 0.5 unit and the cephalin flocculation test was 1 plus in twenty-four hours and 1 plus in forty-eight hours.

X-ray studies revealed many nodules throughout both lungs and hilar regions; these nodules were regarded as metastatic in nature. Although the lesion was diagnosed as a malignant mixed tumor of the parotid gland and there was x-ray evidence of pulmonary metastasis, removal of this mass was considered justified in view of the increasing pain and skin necrosis. At operation removal of the entire mass was not possible since tumor tissue had penetrated the fascial planes of the neck quite deeply with infiltration of the underlying structures. The patient's reaction to the operation was satisfactory. The wound healed without complication and during the remainder of her life there was no evidence of local recurrence. On June 6th the patient was discharged as improved but the facial paralysis remained unchanged.

Pathologic examination of the surgically removed specimen revealed a very large, irregularly nodular tumor mass measuring 15 by 14 by 9 cm. and weighing about 900 gm. Approximately one-half of the mass was covered with purplish white, congested, suffused skin. The nodules ranged in diameter from 1 to 8 cm. and the tumor presented a mottled yellowish to pinkish white color. The tumor was cut with some difficulty and revealed a lobulated, purplish white cut surface with occasional small areas of cystic degeneration. One small nodule was seen, measuring 3 by 4 cm., which was made up of soft, whitish grey homogeneous tissue; a similar soft, pinkish white nodule measuring 4 by 3 by 2 cm. was found on the surface of the tumor. In addition, two light reddish brown nodular pieces of tissue were submitted separately, each measuring approximately 5 by 5 by 2 cm. Upon section their cut surfaces varied from a rather soft, whitish, necrotic consistency to a firmer, more pink appearance.

Microscopic study of the more solid areas showed a connective tissue and chondroid background against which irregularly shaped masses and strands of cells were seen. These cells varied from spindle to polyhedral in shape. The cells appeared somewhat active and exhibited occasional mitoses. (Fig. 2.)

All other sections revealed unquestionable malignancy. In one area dense, almost hyaline, and chronically inflamed connective tissue was invaded and infiltrated by strands of malignant cells which tended to form abortive stratified squamous epithelium, with numerous whorls and epithelial pearls. (Fig. 3.)

Other sections showed large sheets and islands of more or less anaplastic, obviously malignant cells, in which one could detect here and there abortive attempts at cornification. (Fig. 4.) The individual cells exhibited all the usual stigmas of malignancy to an advanced degree. Numerous mitoses were present, many of which were atypical. Many tumor giant cells were observed with large, bizarre, hyperchromatic nuclei. In many fields necrosis was a prominent feature and the surviving tumor cells were arranged perithelially. A definitive diagnosis of malignant mixed tumor of the parotid gland was made.

The patient was readmitted to the Roger Williams General Hospital on October 10th because of pain and swelling in her left middle finger. About six weeks previously her left middle finger had become swollen and painful. The nail sloughed off and the pain and swelling increased.

Physical examination on admission revealed a swollen left middle finger, the distal portion of which, particularly the palmar surface, was

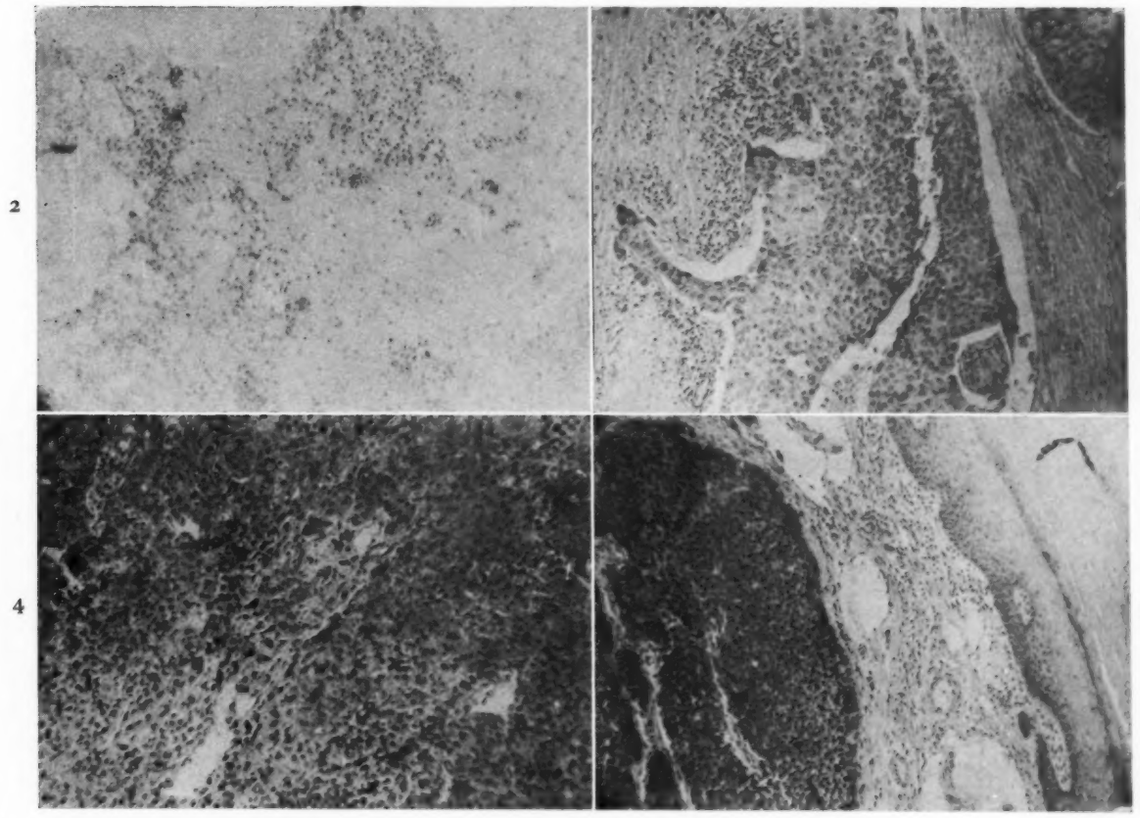

FIG. 2. Masses and strands of tumor cells are set in a chondroid matrix. This pattern is relatively benign. FIG. 3. Masses of malignant cells which exhibit an abortive stratified squamous pattern, invade connective tissue. FIG. 4. There is almost complete anaplasia of the tumor cells. FIG. 5. Anaplastic metastatic tumor cells invade end of finger.

taken up by a soft, fungating, mottled, purplish, reddish yellow growth. There was still pronounced right-sided facial paralysis.

X-ray of the finger disclosed a distal soft tissue swelling without bone destruction or periosteal reaction. Laboratory studies were not contributory.

The distal phalanx of the middle finger was amputated and submitted for pathologic examination. This specimen measured 4.5 by 3 by 2 cm. The distal portion of the specimen, particularly the palmar portion, was taken up by a soft, fungating, mottled, purplish red growth measuring 3 by 3 by ½ cm. The fingernail was absent. Section through the mass revealed soft, easily cut pinkish white tissue, grossly very suggestive of malignancy. Frozen section indicated infiltration with obviously malignant cells occurring in strands and sheets.

Definitive sections disclosed stratified squamous epithelium, beneath which there was a highly anaplastic invasive growth made up of sheets and interwoven groups of large obviously malignant cells. Mitoses were very frequent with many abnormal forms. There was considerable hemorrhage and necrosis. This lesion corresponded in its histologic pattern (Fig. 5) to the original malignant parotid tumor and was considered to be a metastasis of this tumor.

The patient was discharged as improved on October 10th and died approximately one year later. There was no autopsy.

SUMMARY

A case is presented of malignant mixed tumor of the parotid gland. This case showed the following interesting features: (1) the extraordinary duration and size of the primary tumor; (2) histologic pleomorphism of the parotid tumor, ranging from the relatively benign pattern of the so-called "mixed tumor"

of the parotid gland to a picture of complete and aggressive anaplasia (the malignant areas made up only a small portion of the huge mass, however); (3) a very rare metastasis from this primary tumor was encountered in the subcutaneous tissues of the tip of the left middle finger.

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Solitary Renal Cyst Containing Free Stones Simulating Cholelithiasis

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Solitary renal cyst is an uncommon pathologic entity. The presence of free stones within one of these cysts is a rare condition. This pathologic finding is in contradistinction to the multiple small retention cysts of chronic nephritis and polycystic kidney. One of the earliest reports was that of Fabricius Hildanus[2] who described but did not differentiate them from polycystic kidney. In 1876 Laveran[7] made a distinction between single renal cyst and polycystic kidney. Kretschmer,[6] in 1920, collected ninety-nine cases from the literature and added one of his own. Simkow,[11] in a further study, collected 315 cases, and Gutierrez[3] reported ten cases and discussed the subject in detail.

Young[12] stated that of 12,500 urologic cases at the Brady Institute not one of solitary renal cyst was found. However, according to pathologists, small cysts are encountered fairly commonly during postmortem examinations.

The cause of the condition is incompletely understood. Several theories are extant, none fully explaining this unusual pathologic finding. The theory that the cyst has its origin in an embryonic arrest has adherents. Blocking of the tubules with salts and other debris following localized inflammation is considered the basis by many for their development. Hepler[4] was able to produce them in rabbits after fulguration of the collecting tubules and arterial ligation of the area. They may occur at any position in the kidney and are sometimes bilateral. Since the patients reported have been between the ages of seventeen and seventy years, it is presumptive evidence at least that the condition is acquired as the result of pathologic processes of adult life.

Four varieties have been recognized and these have been outlined by Quinby[9] as follows: (1) the simple cyst, thin-walled within the parenchyma and with no anatomic relationship to the kidney pelvis; (2), the cyst or cysts lie within the renal parenchyma, compressing it, and communicate with the renal pelvis; (3), this variety lies outside the kidney but communicates with the kidney pelvis, a modification of, but not a true, hydronephrosis; (4), the hemorrhagic group are those with hemorrhage within the cyst.

The wall of the cyst has been described as consisting of the fibrous capsule of the kidney, connective tissue, atrophic glomeruli and uriniferous tubules. The inner layer may be either pavement or cuboidal epithelium, or transitional type as in the case detailed herein. The contents are serous fluid or blood. When the latter is present, it is the hemorrhagic variety already mentioned. Calcareous deposits in the wall have been reported by Kirwin,[5] Allen[1] and Rudstroem.[10] Free stones within the cavity of the cyst without calcification in the wall is illustrated by Lowsley and Kirwin.[8] Allen[1] also shows one illustration with calcareous deposits within the cyst cavity.

CASE REPORT

The following case is reported in detail because of the rarity of the condition and the diagnostic problem involved.

A fifty-four year old Chinese man was admitted on December 23, 1953, with the chief complaint of pain and swelling in the right knee of three weeks' duration. He also had had frequency and nocturia for several months. Three weeks prior to admission he had injured his knee. He continued to be active but the knee became increasingly swollen and tender and crutches were necessary. Except for fractured legs in 1947 and swollen legs in 1952, his past history was unremarkable.

On physical examination his temperature was 100.8°F. Moderate jaundice of the scleras was present and the right knee was swollen

FIG. 1. X-ray of abdomen: "Multiple biliary calculi in the right upper abdomen."

FIG. 3. Intravenous pyelogram: "This shows normal visualization of the urinary tract including the ureters and bladder.

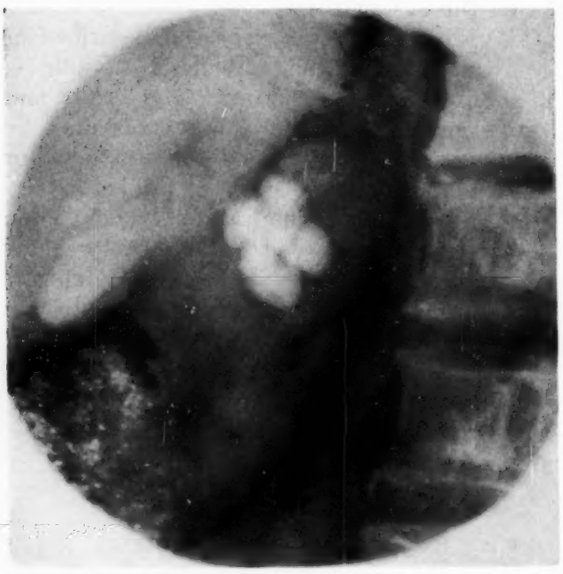

FIG. 2. Gallbladder series: "The gall bladder is normal in size, shape, position, density and motility. A previously noted cluster of six well delineated small calcified densities is located outside, medially and posteriorly to the visualized gall bladder. Whether calcified periduodenal lymph nodes or small gall stones in a secondary gall bladder or in a cholecysto- or cholangiocele remains undetermined."

and tender. Other physical findings were within normal limits.

Laboratory findings revealed the following. The hemoglobin was 8.2 gm. with 3,000,000 red blood cells and 8,600 leukocytes. The sedimentation rate was 144, serum bilirubin 1.2, the alkaline phosphatase 8.2, and the cephalin flocculation negative. Further studies revealed the thymol turbidity to be 0.1, the A/G ratio 3.4 and 2.7, and the non-protein nitrogen 25. Urinalysis revealed a trace of bile and the culture grew Streptococcus hemolyticus.

X-ray of the right knee revealed moderate osteoarthritis with no evidence of fracture. A flat x-ray plate of the abdomen was reported as "multiple biliary calculi in the right upper abdomen." (Fig. 1.)

The knee was aspirated with recovery of a small amount of yellow fluid; 100,000 units of penicillin were instilled into the joint. Culture of aspiration revealed S. hemolyticus. The knee was aspirated three subsequent times with injections of penicillin. Subsequent cultures were negative for any growth. Meanwhile the patient received sulfisoxazole orally daily. During the three weeks of treatment various additional studies were made. The gallbladder series revealed: "the gall bladder is normal in size, shape, position, density and motility. A previously noted cluster of six well delineated small calcified densities is located outside, medially and posteriorly to the visualized gall bladder. Whether calcified periduodenal lymph nodes or small gallstones in a secondary gall bladder or in a cholecysto- or cholangiocele remains undetermined." (Fig. 2.) An intravenous pyelogram revealed the following: "This shows normal visualization of the urinary tract including the ureters and bladder." (Fig. 3.)

On January 22, 1954, the patient was submitted to surgery with a diagnosis of biliary

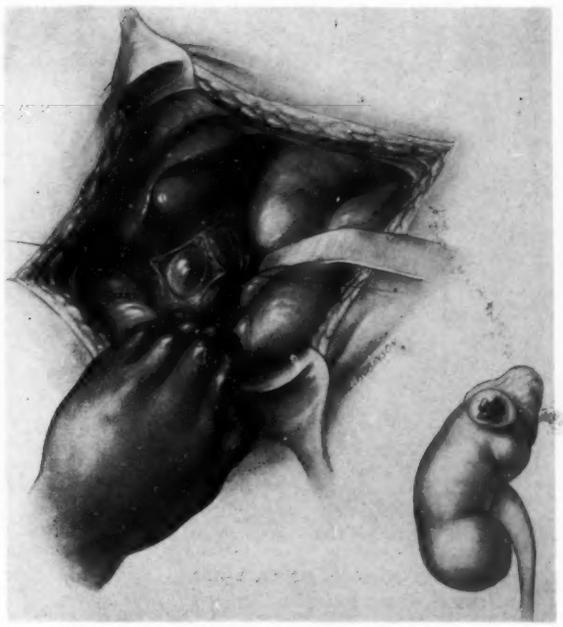

FIG. 4. Artist's interpretation of the operative findings, with site of the lesion and stones within the solitary renal cyst.

disease with a possible double gallbladder, one containing calculi. A right subcostal incision was made. The liver was found to be normal. The gallbladder was normal in size; its wall was moderately thickened, it emptied easily and no stones were palpated. The biliary duct system appeared unremarkable. There were no calcified periduodenal lymph nodes. The possibility of an intrahepatic second gallbladder was seriously considered. Further palpation of the right subhepatic area and right colic gutter elicited a small, hard, movable object retroperitoneally. Accordingly, the hepatic flexure was mobilized downward, the duodenum medially, and the parietal peritoneum over the kidney was incised. At this point a cystic mass at the upper pole of the kidney presented. Within the cyst were the stones previously seen by x-ray. The cyst was unroofed and six brown, very hard facetted stones were removed. The cyst cavity contained about 4 cc. of slightly opaque yellow fluid with no odor. A culture was taken, with no growth. No connection with the kidney pelvis was found. The cyst wall was excised flush with the renal parenchyma. Bleeding was moderate but adequately controlled. Because of the evidence of biliary obstruction on admission, the gallbladder was removed, and the common duct opened and explored, with

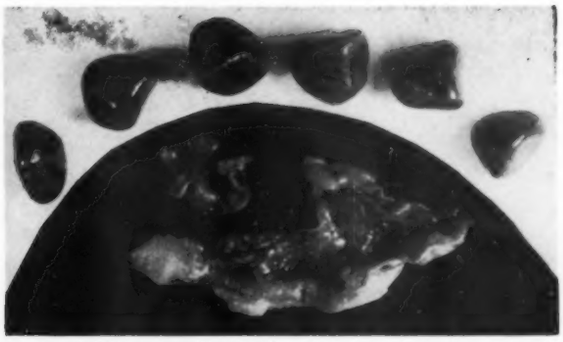

FIG. 5. Photograph of the stones removed and the wall of the cyst.

no stones found. A T-tube was left *in situ*. The abdominal incision was closed and a stab wound for drainage was made in the right flank. (Figs. 4 and 5.) The recovery was smooth and without complications. Postoperative x-rays revealed no residual shadows suggestive of stones.

Analysis of stones revealed the following: "They are shiny, brown smooth calculi; hard to grind, no nucleus. Composed of calcium oxalate, calcium phosphate and urates."

A microscopic section of the cyst wall gave this data: "Greatly altered renal parenchyma lined on its internal aspect by transitional epithelium. Both focal and diffuse lymphocytic aggregations are present in the subepithelial tissue. The cyst wall is of variable thickness and is largely fibrous in nature. In it are seen cystically-dilated tubules containing acidophilic secretion resembling colloid. Variably sized vessels are present showing disuse atrophy and there is extensive interstitial scarring; no glomeruli can be identified. The lesion is unquestionably of pelvic origin." (Fig. 6.)

SYMPTOMS

When the cysts are small there are no signs or symptoms which are diagnostic. When large and situated at the upper pole of the right kidney, a mass may be palpated. If infection is present, there is tenderness in addition to the mass. This combination closely simulates disease of the gallbladder. Pain may be present in the upper abdomen, or posteriorly toward the back. In about one-third of the cases cystitis is present; in another third, chills and fever are evident. Quinby described a patient who was acutely ill; the diagnosis was perforated gallbladder and an emergency operation was performed. A right upper abdominal

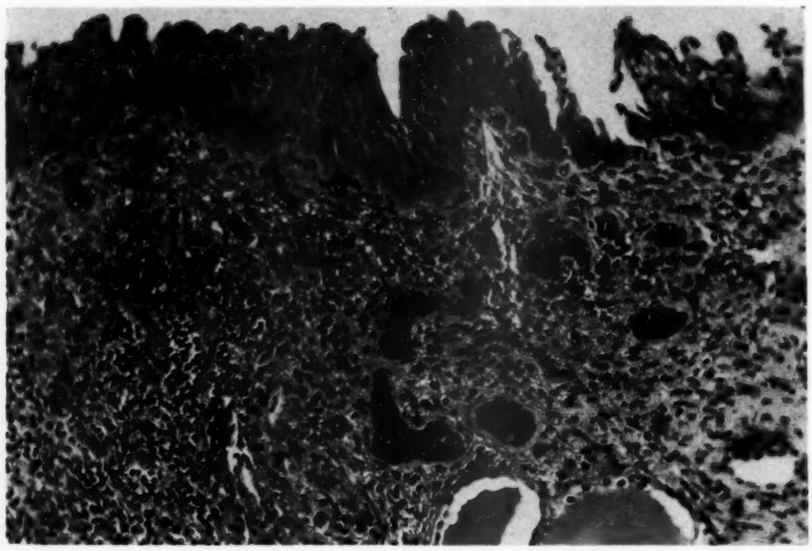

FIG. 6. Photomicrograph of the cyst wall: "Greatly altered renal parenchyma lined on its internal aspect by transitional epithelium. Both focal and diffuse lymphocytic aggregations are present in the subepithelial tissue. The cyst wall is largely fibrous in nature. In it are seen cystically dilated tubules containing acidophilic secretion resembling colloid. Vessels are present showing disuse atrophy and there is extensive interstitial scarring. No glomeruli can be identified."

incision was made and no pathologic disorder of the biliary system was found. A mass was present retroperitoneally. Drainage of a large infected solitary cyst of the upper pole of the right kidney was carried out posteriorly.

In over 50 per cent of the cases the pyelogram shows deformity. Hematuria may be present, but the urinalysis is usually normal. Actually the symptoms are referable to the location and size of the tumor, its pressure effects, its connection with the kidney pelvis, or the presence of hemorrhage or infection. While the diagnosis can usually be made by urologic study, the uncommon case so closely simulates biliary disease that it becomes necessary to resort to surgery for final diagnosis.

TREATMENT

If the lesion is incidentally found, is small and symptomless, no treatment is indicated. Whether definitive treatment is necessary depends upon the size and location of the cyst or cysts, and the presence of infection or hemorrhage. In most instances unroofing and drainage are adequate measures. According to most authorities, nephrectomy is seldom considered necessary. When there is interference with normal function and destruction of the renal parenchyma, nephrectomy may be the procedure of choice. Conservative measures, however, are the first consideration.

SUMMARY

1. Solitary cyst of the kidney with free stones is discussed.

2. Present concepts as to the etiology are mentioned.

3. Symptoms and signs simulating disease of the gallbladder and cholelithiasis are emphasized.

4. The accepted form of treatment is outlined.

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Acute Gangrenous Cholecystitis Secondary to Trauma

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TRAUMA and its effects on all parts of the body have been studied both clinically and experimentally. The literature contains many types of conditions of the gallbladder occurring as a result of trauma (i.e., lacerations, bullet wounds, perforation and enucleation). However, as far as can be seen, acute gangrenous cholecystitis secondary to trauma has never been reported.

Traumatic cholecystitis was reported by Ireneus.[1] McBride's[2] text mentions trauma as a cause of acute cholecystitis. These are the only two recorded cases of acute cholecystitis due to trauma. The subject of this report is a seventy-eight year old man with no previous history of biliary difficulty.

CASE REPORT

N. G., a seventy-eight year old white man, was struck by an automobile on April 13, 1954. The patient was never unconscious. The family history was non-contributory. The past history revealed that the patient had had a ten-year period of sinus trouble; approximately ten years before the present admission the patient had had typhoid fever with pneumonia. Systemic review showed no abnormalities.

Physical examination revealed an oriented, coherent and cooperative white man who looked his stated age. There was a 5 inch laceration over the vertex of the skull extending through the galea to the bone. There were multiple contusions and abrasions over the entire body. The eyes, ears, nose and throat were normal. There was a 1 cm., flat hyperkeratotic area with raised, rolled edges at the mid-portion of the right nasolabial fold. The neck was supple. There was no thyroid enlargement. The heart was enlarged to the left but had normal rate and rhythm. The blood pressure in both arms was 110/70 mm. Hg. The lungs were clear to percussion and auscultation. Abdominal examination revealed no tenderness, spasm, rigidity, masses, fluid wave or shifting dullness. There was no rebound or costovertebral angle tenderness. There was tenderness over both rami of both pubic bones and over the inferior rami of the right ischium. The scrotum was ecchymotic but both testicles were normal to palpation. Rectal examination revealed tremendous spasm but no blood on the finger. There was gross deformity of the left tibia and fibula with tenderness and ecchymosis.

The admission diagnoses were as follows: (1) laceration of the galea aponeurotica, (2) fractures of the pelvis, left tibia and left fibula, (3) multiple contusions and abrasions, and (4) basal cell carcinoma of the face. The laceration was repaired. A cast was applied for the fractures of the tibia and fibula, and the patient was placed at bedrest. About eighteen hours after the accident the patient began to complain of epigastric pain and vomited. Electrocardiographic tracings showed evidence of what might be an acute anterolateral infarction. The patient seemed to respond to medical therapy. However, he continued to complain of epigastric fullness and pain. On the sixth day after admission the patient complained of pain in the right upper quadrant. This subsided rapidly. On the ninth day after admission the scalp wound was dressed and all stitches were removed. On the next day there was an exacerbation of the epigastric pain with radiation to the right upper quadrant accompanied with nausea. On the eleventh day after admission there was an increase in the pain along with distention, rebound tenderness and a positive Murphy sign. A mass was palpable in the region of the gallbladder extending approximately 2 to 3 fingerbreadths below the costal margin in the mid-clavicular line. The white blood count was 22,850. Result of the van den Bergh test was 1.7/0 (indirect/direct). The diagnosis was acute cholecystitis. Conservative measures (rest, antispasmodics, antibiotics, nothing by mouth) were tried. Twenty-four

hours later the patient's condition was worse, with a white blood count of 40,000. Operation was performed because it was believed that perforation was imminent, far outweighing the risk of the patient's coronary status. Under local anesthesia cholecystostomy was performed through a subcostal incision. The gallbladder was found to be markedly distended, thickened and edematous. There was a patchy gangrenous process involving most of the area of the fundus of the gallbladder. No calculi were present. Culture of the peritoneal fluid revealed colon-aerogenes. Culture of the gallbladder contents yielded Aerobacter aerogenes but no evidence of typhoid bacillus. Biopsy taken of the wall revealed gangrene of the gallbladder.

Postoperatively the patient did very well under maintenance digitalis, antibiotics, intravenous fluids and blood. All tests showed improvement. Jaundice disappeared. By the sixth postoperative day the patient's temperature was normal. Twenty days postoperatively open reduction of the tibia and fibula was performed. Twenty-three days after the cholecystostomy the drainage tube was removed. Within two days biliary drainage had ceased. The day before discharge the basal cell carcinoma of the face was excised under local anesthesia. On the thirty-fifth day after admission the patient was discharged to a nursing home.

COMMENTS

One possible cause of disease in this case is that the cystic artery may have contained an arteriosclerotic plaque and the trauma may have caused subintimal hemorrhage and thrombosis, thus leading to gangrene of the gallbladder due to diminished blood supply. Another possibility is that the trauma produced subserosal hemorrhage in the wall of the gallbladder with subsequent necrosis. The absence of a previous history, the absence of calculi and the onset of the disease immediately following trauma in a seventy-eight year old man would suggest that the first explanation is the more favorable one.

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Traumatic Diaphragmatic Hernia*

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TRAUMATIC diaphragmatic hernias are being encountered with increasing frequency in consequence of highway and other violent accidents. Not uncommonly the trauma to the diaphragm may be initially overshadowed by severe concurrent injuries. The diaphragmatic hernia may then escape detection until some later date when visceral strangulation occurs. The converse is also true. A patient may present himself with symptoms of obstruction of the gastrointestinal tract; and unless care is taken to elicit a history of previous trauma, the diagnosis of diaphragmatic hernia may not be entertained. The two cases to be presented illustrate these pitfalls.

The review of a large reported series[2,9,11] reveals that penetrating wounds are the most frequent etiologic factor, with stab and gunshot wounds predominating. Shell fragment wounds are prevalent in wartime. Closed trauma due to thoracic contusion, or crushing abdominal injury, is becoming of greater importance as high-speed automobile accidents increase. A fall from a height rarely produces diaphragmatic injury. Inflammatory necrosis of the diaphragm secondary to a subdiaphragmatic abscess or an intrathoracic drainage tube may be a cause of a ruptured diaphragm. Postoperative herniation following incision of the diaphragm has been reported.[11]

The embryology and anatomy of the diaphragm are of significance in the congenital and acquired types of hernia, but are of little value in predicting the location of a traumatic defect which need not conform to the sites of potential weaknesses. Only about five per cent of traumatic diaphragmatic hernias occur on the right due to the splinting action of the adjacent liver but, when encountered, these right-sided defects are usually very large, allowing the liver and sometimes the colon to enter the right thoracic cavity. The left diaphragm is involved in the great majority of cases and subsequent discussion will be limited to left-sided lesions only.

Herniation does not always occur immediately after injury. Of the twenty-eight cases reported by Hughes et al.,[11] hernias developed in twenty-three immediately after injury while in five cases it appeared at a later date. When the diaphragm is injured the ensuing events are unpredictable and dependent upon various factors which include the site and size of the defect, the relative position of the viscera, the interplay of positive and negative pressures, and subsequent stresses and strains. For this reason Carter et al.,[3] in a masterful presentation of the subject, divided the symptomatology and diagnostic signs into three phases. First, that immediately following the accident; second, that of the interval; and third, the phase of obstruction and strangulation. During the first phase concomitant injuries may obscure that of the diaphragm which at the time may be relatively insignificant. This is especially true in small penetrating wounds in which hemothorax or damaged thoracic and abdominal viscera predominate. The diaphragmatic defect may then be plugged by omentum. This is a frequent occurrence and may prevent visceral herniation for the moment; however, the stage is set for future trouble since the margins of the defect in the diaphragm are prevented from healing, rendering the site vulnerable to future stresses. The closed or non-penetrating diaphragmatic injury results from a sudden compression of the thorax or abdomen producing a bursting force. The resulting rent is usually a large one, extending radially from the region of the esophageal hiatus across the dome to the costal attachments. At times the costal fibers may even be torn away from the thoracic wall. Such a severe injury should be expected to produce a huge herniation with immediate symptoms of respiratory and cardiac embarrassment, pain and shock.[3]

* It is with regret that this article as it originally appeared in the December 1955 issue contained an error in continuity. We are therefore rerunning the article in its proper sequence.

† Present address: Hazard, Kentucky.

Upon physical examination, dullness or areas of tympany will be present over the left lower thorax and there may be a shifting of the mediastinum to the right. The distinctive physicial sgns listed by Gibson[7] over twenty-five years ago are: (1) diminished excursions of the thorax; (2) impairment of resonance; (3) the absence of the Litten diaphragm phenomenon; (4) adventitious sounds in the chest; (5) cardiac displacement and (6) circulatory collapse. Patients who recover from the acute injury may subsequently have few or no symptoms if the diaphragmatic defect is occluded by omentum or by a solid viscus such as the spleen on the left or the liver on the right. The partial protrusion of a portion of stomach or a loop of bowel, as long as it remains unobstructed and its blood supply uncompromised, produces only mild symptoms of a varying nature. Thus, during the interval phase diagnosis is seldom made except by persistent and exacting roentgenographic examinations. After a very variable period of time, which may be from one day to twenty-three years[15] but is usually within the first two or three years of injury,[3] the phase of obstruction or strangulation is reached. This phase is almost always initiated by some relatively minor injury or episode causing increased intra-abdominal pressure. The onset is sudden with severe pain in the left hypochondrium or lower thorax. Radiation of pain to the neck and shoulders is especially suggestive of diaphragmatic involvement. When small or large bowel is obstructed, vomiting, progressive distention, hyperperistalsis and obstipation develop. If the stomach alone is involved in the strangulation, excessive vomiting and hematemesis may be the only significant early symptoms. Recovery of bloody fluid by thoracentesis strongly suggests visceral strangulation.

Roentgenographic findings depend upon the size and the components of the visceral herniation and the amount of pleural fluid present. The routine upright films may show merely a picture resembling an abnormally elevated diaphragm or eventration.[5] On the other hand, with a large hernia extensive changes consisting of right mediastinal shift and a left thoracic cavity filled with abnormal densities and gas pockets (occasionally with distinct intestinal patterns) may be revealed. Studies with barium by mouth or by enema are frequently necessary to identify the viscera displaced into the thorax, or the site of obstruction. Associated hemothorax is to be anticipated in both the initial phase after injury and when strangulation occurs. It may obscure the herniated structures, and films in both the upright and recumbent positions are essential in arriving at a diagnosis. The differentiation of pyopneumothorax or pleural effusion may also have to be made, especially in late cases.[15]

The treatment of traumatic diaphragmatic hernia is surgical repair of the diaphragmatic defect. The transthoracic approach gives superior exposure but should be planned for conversion into a thoracoabdominal incision should this be necessary. Using this approach, concomitant intrathoracic trauma can be adequately dealt with in cases of fresh injury. When surgery is performed at a later date, adhesions intimately binding the omentum and herniated viscera to the margins of the defect or to the intrapleural structures can be well visualized and safely severed through a transthoracic approach. When strangulation is discovered the incision may be extended across the costal margin into the upper abdomen to afford adequate exposure for abdominal exploration or indicated resection. Phrenic nerve crush not only facilitates the repair of the diaphragm but also minimizes recurrence in the postoperative period. This procedure can be performed through the thoracotomy without resorting to a secondary operation in the neck.

CASE REPORTS

CASE 1. E. B., Jr., a thirty-one year old man, was admitted on December 16, 1953, to the Hospital of the Biloxi V. A. Center with abdominal pain, nausea and vomiting of forty-eight hours' duration. He had been in excellent health until approximately 5 P.M. on December 14, 1953, when he stumbled while wheeling a heavy wheelbarrow filled with cement. He caught himself without falling but experienced a sharp pain in the left hypochondrium. This pain was constant, did not radiate and gradually increased in severity. Three hours after the onset he attempted to eat but this induced nausea and vomiting. Subsequently the pain persisted, and on the day of admission it became cramping and increasing abdominal distention was noted. Obstipation from the onset was not relieved by a cathartic or enemas. Frequent vomiting

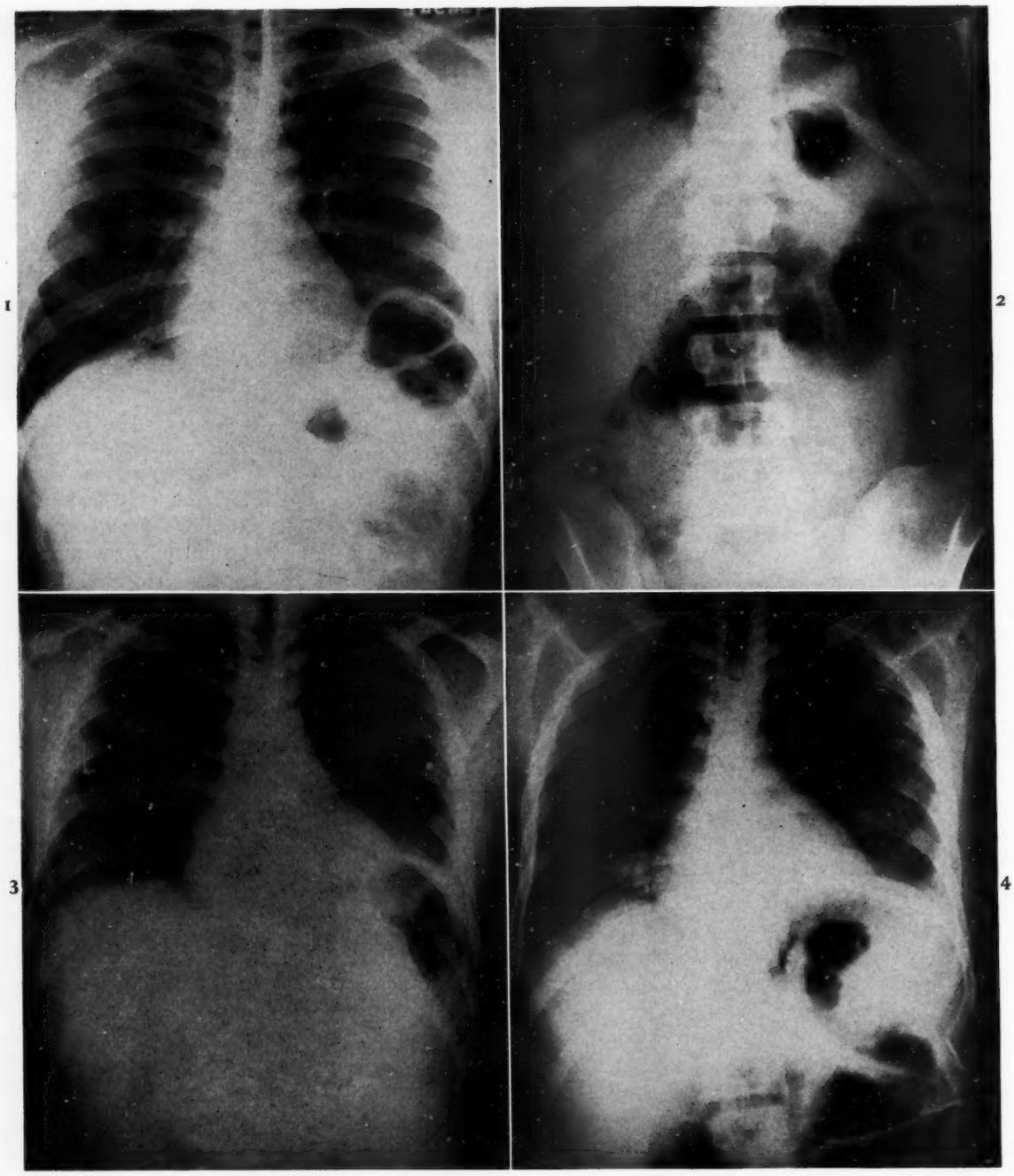


FIG. 1. Case 1. Incarcerated loop of colon protruding through left diaphragmatic defect.

FIG. 2. Same case. Distended colon proximal to obstructing herniation.

FIG. 3. Same case. Minimal findings shortly after a knife wound of the left chest eighteen months previously.

FIG. 4. Same case. Roentgenogram following reduction of the left hernia and repair of the defect. The left diaphragm is elevated due to phrenic nerve crush.

continued and not even water could be tolerated. The pain was more severe upon lying down and partial relief was obtained in the upright position. At no time was pain referred to the shoulder.

On admission the physical examination revealed an extremely well developed and well nourished young man who was cooperative and well oriented. He appeared acutely ill, anxious and complained of pain in the left upper abdomen. The temperature was 37° C., pulse 80, respiration 20 and blood pressure 128/78. The entire physical examination was essentially within normal limits with the exception of exquisite tenderness in the left upper quadrant of the abdomen, splinting of the left thorax and a fullness in the epigastrium. There was no muscle spasm or rebound tenderness. Peristaltic sounds were infrequent. Rectal examination was negative except for the presence of internal hemorrhoids.

The initial hemogram revealed a white blood count of 10,600 with 77 per cent neutrophils. The red blood count was 4,500,000 with 14.3 gm. of hemoglobin. Urinalysis and the blood Kahn reaction were negative. A roentgenogram of the thorax taken in the upright position on admission (Fig. 1) was reported as follows: "There is an abnormality in the left base. The outer two-thirds of the left diaphragm shows marked doming. A gas filled loop of the colon fills this area. The wall above the gas filled loop is rather thicker than that expected if this consisted entirely of the colonic wall. This domed area fuses into the diaphragm on the medial side in a wide obtuse angle. The general appearance is more that seen with congenital defect than of an acute herniation of the colon through a defect in the diaphragm." Further roentgenographic views in the upright and supine positions revealed moderate gaseous distention of the transverse colon (Fig. 2) and small collections of gas and fluid in loops of small bowel in the right lower quadrant.

Following these initial x-rays, a Cantor tube was inserted into the stomach and another roentgenogram was obtained. This revealed that the stomach was entirely within the abdominal cavity. Diagnosis of traumatic diaphragmatic hernia with an obstruction of the colon was suspected. The patient was given intravenous fluids, started on gastric suction and the left phrenic nerve was injected at a point just above the clavicle with 4 cc. of 1 per cent xylocaine. This produced considerable relief of pain and the patient fell asleep.

On the following morning the patient again complained of pain. Further roentgenograms revealed that obstruction had not been relieved and there was increasing distention of the transverse colon and distal small bowel loops. At this time an old thoracic roentgenogram was discovered in the files under this patient's name. Upon further questioning, the patient recalled and admitted having had a knife wound of the left chest eighteen months previously. The knife had entered the left chest in the ninth interspace at the anterior axillary line, resulting in a wound which sucked and bubbled with respirations. He was taken to a small community hospital where he remained in bed for two days, receiving no specific treatment or diagnostic studies. He was then discharged and returned home. Pain in the left chest, especially with respiratory efforts, persisted for approximately one week. Two weeks after his injury he was seen in the admission department of this hospital. At that time he complained only of having had the stab wound but all his symptoms had subsided. A single postero-anterior roentgenogram of the thorax was made which was reported as showing "slight elevation but normal contour of the left diaphragm with slight pleural thickening and blunting of the left costophrenic angle." (Fig. 3.) There appeared to be no reason for admission and he subsequently had no further difficulty for eighteen months.

Re-examination of the patient in the light of this history revealed an almost imperceptible, 2 cm. scar in the left ninth interspace along the anterior axillary line. He was then taken to the operating room and under endotracheal anesthesia the left thorax was entered through the bed of the tenth rib in order to avoid the area of the previous knife wound. Exposure was excellent, revealing a huge mass of omentum and a large distended loop of colon protruding through a circular, 5 cm. defect in the anterior portion of the left diaphragm. The omentum was densely adherent to the edges of the defect and a long, thick tongue of omentum extended upward and was firmly attached to the anterior thoracic wall immediately beneath the scar of the old knife wound. The adherent omentum was dissected free from the lateral edges of the defect and the diaphragm was opened widely. The phrenic nerve was then

crushed with a hemostat just above the diaphragm. The incarcerated loop of colon, identified as the splenic flexure, was somewhat dusky but rapidly assumed a normal color upon opening the thickened fibrotic edge of the diaphragmatic defect. No other abnormality was found upon exploration of the upper abdominal organs through the opened diaphragm. The repair of the defect was accomplished by overlapping the posterior and anterior leaves employing two rows of No. 00 silk mattress sutures and a row of simple pleuralizing sutures at the edge. The chest cavity was irrigated with saline, the lung completely expanded and the chest tightly closed in layers with interrupted sutures of Nos. 00 and 000 silk. The patient received 1,000 cc. of whole blood during the procedure.

Postoperatively, for twenty-four hours the patient was treated with constant gastric suction. He then began to expel gas by rectum and was started on a liquid diet which was rapidly graduated to a full diet. Penicillin and streptomycin were administered intramuscularly. A low grade fever occurred during the first five postoperative days, but otherwise he had an uncomplicated course. Postoperative roentgenograms (Fig. 4) showed a high left diaphragm and a right basal atelectasis which cleared rapidly following forced coughing. A left diaphragmatic pleural reaction was noted during the first postoperative week, but this gradually cleared. He was subsequently discharged as well without further complication.

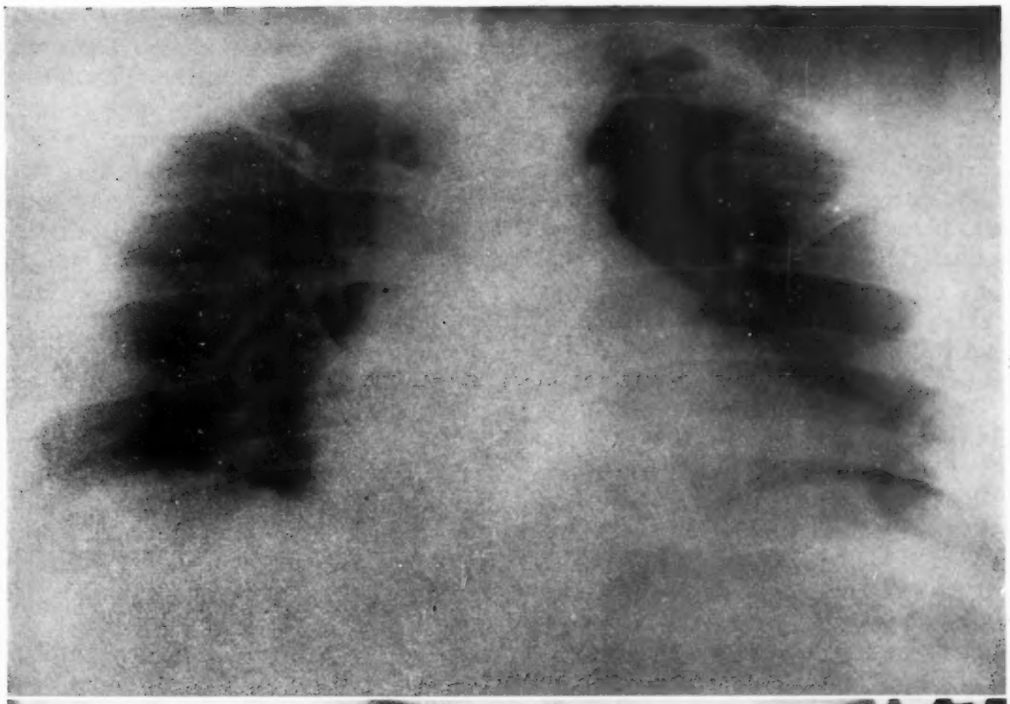
Comment. This case illustrates many of the characteristic features following a penetrating diaphragmatic wound. The sequence of events is typical and in fact has a repetitious ring after reviewing case reports of similar injuries. The progressive nature of the lesion, the delayed abrupt onset of symptoms and the propensity of the omentum for plugging abnormal openings is well demonstrated.

CASE II. L. G., a twenty-eight year old hospital attendant, was admitted at 5 A.M. on October 3, 1952, by ambulance to the hospital of the Biloxi V. A. Center. Approximately two hours previously he suffered multiple contusions and fractures when the car he was driving collided with a large trailer truck. The patient admitted having had several drinks before the accident occurred. He was not rendered unconscious.

Physical examination revealed an obese youth who appeared to be in shock. He was pale and breathing rapidly. The blood pressure was 60/40, the pulse weak and rapid, and the respiratory rate 30 per minute. He responded to questioning and there was a strong odor of alcohol on his breath. The pupils were equal, reactive to light and accommodation, and there were no obvious neurologic defects. Many superficial abrasions and contusions were noted over the left chest and abdomen, together with tenderness over the lower left chest posteriorly in the region of the ninth to eleventh ribs. The chest excursions were equal bilaterally and auscultation revealed normal breath and heart sounds. The abdomen was soft and no definite tenderness could be elicited over the left upper abdomen. No surgical scars or abdominal hernia were present. There was a closed fracture of the mid-shaft of the left humerus and an open fracture of the left tibia and fibula at the junction of the lower and middle thirds.

The patient voided voluntarily and the specimen was essentially normal and free of blood cells. The hemogram revealed 3,700,000 red cells per cu. mm., a hemoglobin of 11.9 gm. per cent, 16,700 white cells with 91 per cent neutrophils and 9 per cent lymphocytes, and bleeding and coagulation times within normal limits. Because of the extensive injuries only a portable bedside roentgenogram of the thorax was obtained with the patient supine in the shock position. This was considered to be within normal limits for such an obese man. (Fig. 5.)

Immediate treatment consisted of administration of 500 cc. of plasma followed by 500 cc. of compatible blood. The foot of the bed was raised on shock blocks and he was given codeine for pain and placed in an oxygen tent. The blood pressure gradually rose to 120/80 but the pulse rate remained at 110. Six hours after admission the patient was taken to the operating room and the compound fracture of the left leg was débrided, reduced and fixed with a Lane plate and screws under general anesthesia of cyclopropane, oxygen and ether. A full plaster cast was applied. A hanging plaster cast was then applied to the left arm. The operative procedure was tolerated well with very little variation in blood pressure. A second unit of whole blood was administered during the operative procedure. After surgery was completed and the patient was beginning



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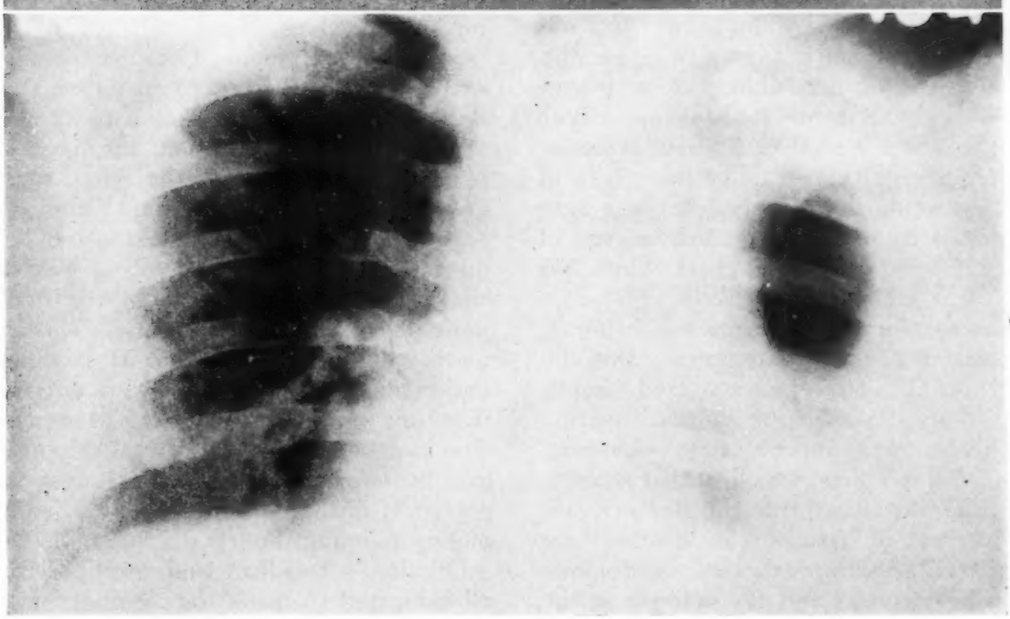

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FIG. 5. Case 11. Roentgenogram of chest with patient in a supine position shortly after injury.

FIG. 6. Same case. Massive herniation of abdominal viscera into left thoracic cavity twenty-four hours later. Gas pocket lies within large bowel loop.

to respond, a rapid pulse was noted and transitory cyanosis followed by flushing of the face was observed. He apparently responded to intratracheal suction and was removed to the recovery room. Five hours after surgery his blood pressure was recorded at 126/60 and the patient appeared to be progressing satisfactorily. Eight hours after surgery the pulse rate had increased to 128 and respirations to 30; however, he slept soundly except when aroused and no other symptoms were noted. Sixteen hours after surgery progressive dyspnea was noted. At that time his temperature had leaped to 104°F. rectally, his pulse was 130,

respirations 36, and blood pressure 150/90 mm. of mercury. He complained of left shoulder pain which was attributed to his fractured left humerus. He presented a picture of severe respiratory distress with diminished respiratory excursions on the left side. There was, however, no intercostal retraction. Although the chest percussion note was considered resonant bilaterally, breath sounds were absent on the left side and the heart was displaced to the right. His abdomen appeared distended and no peristaltic sounds could be heard. His extreme obesity however, made examination difficult from the beginning. His red blood count had fallen to 3.9 million with a hematocrit of 38. The urine was scanty, dark brown in color, and contained 2 + albumin, numerous red blood cells and a few granular casts and white blood cells.

The patient was placed in an oxygen tent, blood transfusions were started and an emergency portable roentgenogram of the chest was obtained. This revealed complete opacity of the entire left lung field. A film of the chest was then obtained with the patient sitting upright. This showed the left hemothorax to be opaque except for two areas of radiolucence which remained constant in spite of different positioning of the patient. (Fig. 6.) The possibility of massive herniation through the left diaphragm was suggested by one observer but in view of the "negative" admission chest film this diagnosis was considered unlikely. The consensus was that he had a pneumohemothorax, and thoracentesis was attempted. Initially no blood or air could be recovered. Later, another thoracentesis with much maneuvering of the needle was successful in obtaining 350 cc. of fresh red blood which clotted rapidly. A Levin tube was passed into the stomach and a small amount of barium was injected into the tube. Roentgenograms showed the stomach in the left hemothorax and gas shadows in the left chest with the typical haustral markings of large bowel loops. Immediate surgery was advised but by this time the patient's condition was precarious. Extreme respiratory embarrassment developed in spite of oxygen therapy. Multiple blood transfusions failed to alter the hemoglobin or hematocrit values. Extreme shift of the mediastinum to the right was followed by rapidly developing pulmonary edema and the patient died at 3:05 P.M. on October 5, 1952, fifty-eight hours after admission.

Postmortem examination was obtained with the following gross findings. There was blood in both thoracic cavities. The left thoracic cavity contained the entire omentum, stomach, transverse colon and spleen. These viscera were herniated through a wide open rent which began just to the left of the esophageal hiatus and extended to the lateral costal attachment of the diaphragm. The spleen was ruptured at its hilum and two lacerations were present on its anterior surface. The left lung was compressed, atelectatic and displaced high in the left thoracic cavity. There was extreme displacement of the mediastinum and a large hematoma, apparently arising from torn periesophageal veins, was present about the mediastinal structures. Blood extended into the right thoracic cavity through a tear in the right mediastinal pleura. The right lung was congested. No ribs were fractured. The peritoneal cavity was filled with blood. Although minimal ecchymosis extended into the left perirenal fat, no injury was found in the kidneys themselves. The fractures of the extremities were noted. Complete examination and sections of the organs, including the brain, disclosed no other remarkable abnormality.

Comment. In retrospect, the most unusual feature of this case was the initial roentgenographic findings of a "normal" thorax. More remarkable, in view of the extensive diaphragmatic defect found at necropsy, was the fact that this film had been obtained with the patient in a supine position with shock blocks under the foot of the bed. It is difficult to understand why herniation did not occur at that time if such a huge defect had been present. One can only assume that the lesion was at first incomplete and that subsequent stresses, especially during anesthesia, succeeded in completely splitting the left diaphragm.

Obesity, alcoholism and multiple fractures all conspired to mask the meaning of obvious clinical and laboratory findings. Nevertheless, all factors in the postoperative course pointed to continuing hemorrhage as well as cardiac and respiratory embarrassment. The wisdom of persisting in attempts to aspirate the thoracic cavity in face of an obscure condition is indeed questionable. Splenic rupture accompanying diaphragmatic hernia is very uncommon but has been reported.[6] This combination predicates a contusion as well as a compression type injury. In this case no rib fractures were present

in the left lower chest, a factor which often assists greatly in making the diagnosis of splenic rupture.

SUMMARY AND CONCLUSIONS

1. Traumatic diaphragmatic hernia is the type chiefly responsible for the complications of alimentary canal obstruction, strangulation and acute massive herniation.

2. A brief review of the subject is presented with special emphasis placed upon the progressive pathologic processes which peculiarize this disease.

3. The intracorporal events following diaphragmatic injury are unpredictable. There may be a time lapse of hours to many years before the symptoms and signs of hernia appear.

4. Cognizance of these characteristics will increase the index of suspicion and so facilitate earlier diagnosis of diaphragmatic hernia due to trauma.

5. Two new cases, one resulting from a penetrating wound and the other from a closed injury, are reported.

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A Highly Fatal Type of Internal Hernia

ALBERT WOLFSON, M.D., *Denver, Colorado*

EVERY patient who has had gastric surgery resulting in gastroenterostomy has a potential hernia through which massive herniation of small bowel can occur.

Fortunately, herniation of small bowel after gastroenterostomy is not a common complication. When it has occurred it has been fatal in most instances. It is interesting to note that in none of the cases reported has the diagnosis been made preoperatively.

CASE REPORT

A sixty-three year old man was admitted with the complaint of knife-like abdominal pain of five hours' duration.

He had been entirely well until he became acutely ill with generalized abdominal pain which was sharp and unremitting. He did not suffer from nausea, vomiting or diarrhea. Within one-half hour the pain became colicky, still generalized, but somewhat more severe in the lower abdomen. Self-induced vomiting offered only slight relief.

Systemic review did not elicit any other significant complaints. The patient had been working and felt entirely well until the onset of this present illness.

His past history revealed that he had had a perforated peptic ulcer twenty years previously which was treated surgically by simple closure. In 1948 he had had subtotal gastric resection with anterior anastomosis because of a persistent gastric ulcer. The ulcer was found to be benign. Since then he had been free of any gastrointestinal complaints.

Physical examination revealed that the patient was acutely ill. Significant findings were limited to the abdomen which was thin-walled and flat except for some slight distention in the lower portion. There was generalized tenderness to palpation, most severe in the lower abdomen. There was no rigidity, rebound tenderness, flank tenderness or masses. There was a small hernia in an upper abdominal incisional scar which was not tender and was free of any abdominal content. There was no dullness to percussion, and auscultation revealed no peristaltic activity.

X-ray examination of the abdomen included an upright chest and a scout film. There was no free air under the diaphragm and no characteristic gas patte n. The white blood count was 13,200 with a slight shift to the left.

It was our belief that this case presented an acute surgical emergency. The preoperative diagnoses were as follows: (1) perforation of a marginal ulcer at the site of gastrojejunostomy; (2) high intestinal obstruction should be considered.

Exploratory laparotomy was carried out within a few hours after admission. The operative finding consisted of a massive herniation of ileum behind the gastrojejunostomy as illustrated in Figure 1.

After the hernia was reduced, the circulation in the incarcerated bowel was immediately restored. The bowel appeared viable and intact throughout. The patient had a benign postoperative course. In the two years following surgery the patient has been free of gastrointestinal complaints.

COMMENTS

Those defects forming hernial rings will undoubtedly be an increasing cause of intestinal obstruction in future years due to the steadily growing number of patients having gastric surgery.

Cannon and Weeks[1] have suggested a simple method for obliterating the formed defect in posterior gastroenterostomy. They believe that closure of this hernial ring is as important as the accepted practice of closing the rent in the transverse mesocolon in posterior gastroenterostomy. (Fig. 2.) Using No. 000 gastrointestinal catgut and beginning at the ligament of Treitz, they suture the proximal loop of jejunum to the transverse mesocolon. Their experience with more than fifty cases has been without complications attributed to closure of this ring.

Highly Fatal Type of Internal Hernia

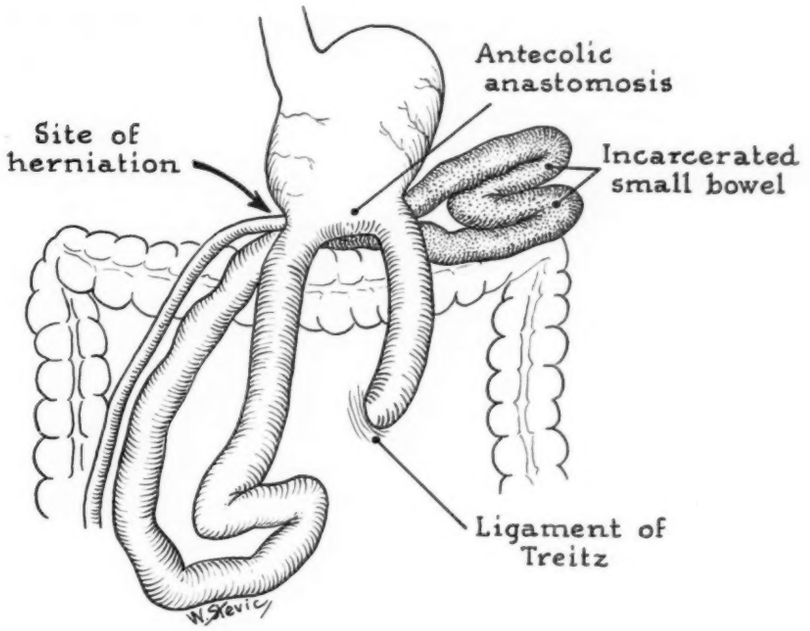

FIG. 1. Diagrammatic illustration showing the internal hernia.

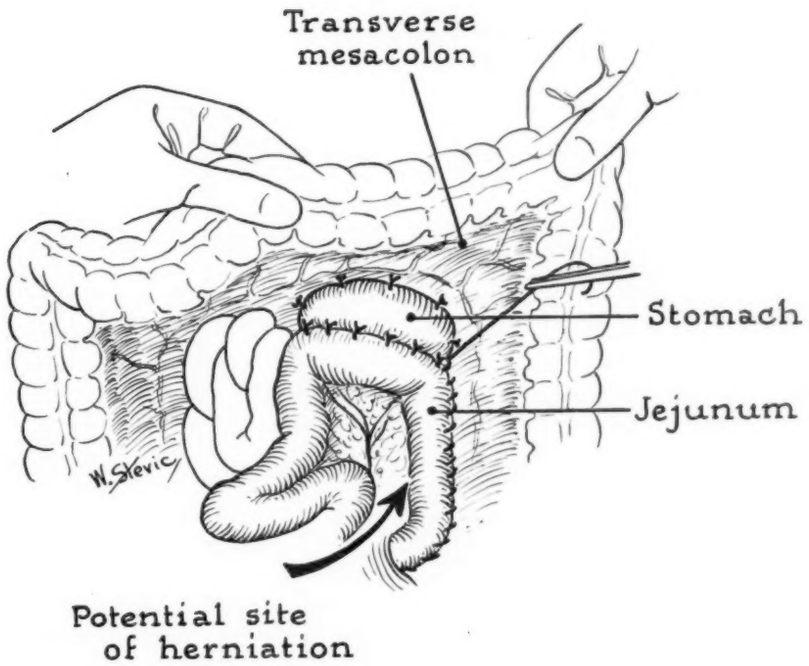

FIG. 2. A method of closing the internal hernial ring in posterior gastroenterostomy. (Modified from CANNON, J. A. and WEEKS, W. H.[1])

The only method we find in the literature that is designed to close the defect in an anterior gastrojejunostomy is reported by Stammers.[2] He suggests suturing the appendices epiploicae of the transverse colon to the efferent loop of gastroenterostomy.

SUMMARY

A case report of an uncommon and highly fatal type of internal hernia is presented. Several methods are reviewed which are designed to obliterate the internal hernial ring either as a prophylactive measure or following herniation.

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We recommend:

A Textbook of Medicine. Edited by Russell L. Cecil, M.D., SC.D., Robert F. Loeb, M.D., SC.D. Associate Editors: Alexander B. Gutman, M.D., PH.D., Walsh McDermott, M.D., Harold G. Wolff, M.D. Ninth edition, 1,786 pages, illustrated (some in color). (Anyone in a surgical specialty should have a basic and fundamental knowledge of medicine as a whole. This work is just the text to fill that need.) Philadelphia, 1955. W. B. Saunders Co.

Peptic Ulcer. Diagnosis and Treatment. By Clifford J. Barborka, M.D., M.S., D.SC. and E. Clinton Texter, Jr., M.D. 290 pages, 33 illustrations. Boston, 1955. Little, Brown & Co. *Price* $7.00.

Traumatic Hernia

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From the Surgical Service of the Newell Hospital, Inc., Chattanooga, Tennessee.

HERNIA resulting directly from non-penetrating trauma to the abdominal wall is extremely rare. Watson[6] states that probably not more than one hernia in 10,000 is traumatic in origin, and Moorehead[4] writes that he never saw a case of genuine traumatic hernia due to a non-penetrating injury. A few cases may be found in the literature, each representing an individual case report.[1,2,3,5]

The following criteria have been suggested as essential for the diagnosis of a true traumatic hernia:[2] (1) There must be immediate descent of the hernia following trauma. (2) There must be severe pain in the region of the injury. (3) There must be a certain degree of prostration. (4) Symptoms must be severe enough for the patient to call attention to it within twenty-four hours. (5) There must have been no hernia previous to the trauma. (6) There must be evidence that the trauma was adequate. In addition, Meade and Murray[3] believe that no preformed sac must be present. The peritoneum may or may not be intact, however, and the hernia may present beneath the skin or beneath one of the fascial layers of the abdominal wall.

The rupture of the muscles and fascia of the abdominal wall may occur as a clean sharp tear, but there is always a certain amount of hemorrhage in the adjacent tissues. Usually, visible evidences of trauma such as abrasions and ecchymoses are present on the skin.

Besides producing rupture of the abdominal wall, trauma may also damage the underlying viscera, and omentum, bowel or mesentery may be injured. Severe hemorrhage may occur, and devascularization of a segment of bowel result if important vessels are torn. Also, the bowel may become incarcerated or strangulated in the hernia if it is not repaired. Injury to the bladder is possible, but it is not likely that one of the solid viscera would be damaged by a non-penetrating injury that produced a hernia, because of their more protected position.

Exploration and repair of the hernia should be performed as soon as possible after the injury. Internal bleeding may require control and devitalized tissue may need resection. Concomitant injuries may demand attention before or after laparotomy, and each case must be handled according to the individual requirements.

CASE REPORTS

C. L., a fourteen year old boy, was admitted to Newell Hospital on June 6, 1953. He had been riding a motor bike which was struck by a truck. The handlebar was turned sharply, and the end thrust into the left lower quadrant of the abdomen. The boy was thrown to the pavement and injured his right wrist. He was brought to the hospital immediately.

Examination revealed a well developed and nourished boy in acute distress. The blood pressure was 100/60, and the pulse 80 per minute. There was an obvious deformity of the right wrist. The abdomen presented an abrasion over the left anterior superior iliac spine and adjacent left lower quadrant. Exquisite tenderness was present over the lateral edge of the left rectus muscle below the umbilicus, and on coughing or straining, a distinct bulge measuring 3 by 5 cm. was noted in this area. Further palpation readily demonstrated a defect here. The rest of the abdomen was soft, and normal peristalsis was present.

The leukocytes numbered 12,750 per cu. mm., and 75 per cent were polymorphonuclears. The urine presented no abnormalities, and x-ray examination of the abdomen revealed no free air, distended loops or fluid levels. X-rays of the right wrist demonstrated an oblique fracture into the epiphysis with posterior and medial angulation.

The wrist was splinted, analgesics and intravenous fluids were administered, and the blood pressure and pulse were stabilized. General

anesthesia was administered and a longitudinal incision was made directly over the defect. On dividing Scarpa's fascia, the peritoneum bulged into the wound through a rupture of the muscle layers at the linea semilunaris. This was about 4 inches in length with equal components above and below the semilunar fold of Douglas. A small amount of blood had extravasated into the adjacent tissues. The peritoneum was opened, and omentum and sigmoid colon were found projecting through the muscle layers. Although there was a hematoma in the sigmoid mesocolon, there was no active bleeding, the bowel was viable and thorough exploration revealed no other injuries. The layers of the abdominal wall were readily approximated with interrupted silk sutures.

The fracture of the forearm was reduced and a short arm cast was applied.

The patient made an uneventful recovery and was allowed to leave the hospital on the seventh postoperative day. The fracture healed well and in good position, and the abdominal wall has remained strong.

COMMENTS

The rare occurrence of a true traumatic hernia may be explained by the specific type of injury that produces this lesion. All cases reviewed and the present case exhibit one feature in common. In each instance the hernia was produced by a sharp blow of a blunt object over a point of weakness or fixation of the abdominal wall. The object must strike with enough force and over a sufficiently small area to produce rupture of the muscle, and at the same time be dull enough not to break the skin. Previously reported cases have been produced by a plow handle, wagon tongue, wheelbarrow handle, and handlebars of a motorcycle or bike. All of these objects are blunt with a relatively small end. Larger objects disperse their force over a greater area so that rupture does not occur, while smaller objects striking with sufficient force tear the skin in addition to the muscles. All of the cases found in the literature have occurred in the inguinal regions or low abdomen, and the rupture has usually occurred adjacent to a point of fixation or at a point of weakness of the flat muscles. Thus, a set of rather unique circumstances appears to be necessary for the production of a true traumatic hernia due to a non-penetrating injury, and the lesion may be expected to remain a relatively infrequent one.

Although a certain degree of shock occurs with a traumatic hernia, the real gravity of the injury is dependent upon the damage to the underlying viscera. Exploration should be performed promptly, and the hernia can be repaired easily by approximation of the severed tissues.

SUMMARY

1. True traumatic hernia due to a non-penetrating injury is rare.

2. Such a case is presented. The treatment of choice is prompt repair. Damage to underlying viscera determines the seriousness of the injury.

3. The lesion probably will continue to be seen infrequently due to the exceptional circumstances necessary for its production.

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BOOK REVIEWS

Human Limbs and Their Substitutes. By Paul E. Klopsteg, PH.D. and others. 844 pages, illustrated. New York, 1954. McGraw-Hill Book Company, Inc. *Price* $12.00.

This splendid work is by thirty-two authors, specialists in their fields. It was prepared under the sponsorship of the Advisory Committee on Artificial Limbs, The National Academy of Sciences, and the National Research Council, in summary and correlation of a research program for the Department of Medicine and Surgery, U. S. Veterans Administration, and for the Office of the Surgeon General, Department of the Army.

We know that science and technology have enabled man to increase the natural powers of his body. Compare the wooden stump leg with a modern prosthesis. The substitutes are the function of machines. This book reveals how scientists have extended that function by augmenting the powers of those whose bodies have been crippled by injury and disease.

This book is the result of an eight-year study presenting conclusions of engineering and medical studies of the human extremities and application of the data to the design and fitting of artificial limbs, and to the care and training of amputees. It is a work which deserves the highest praise.

Diseases of the Liver. By Mitchell A. Spellberg, M.D. 646 pages, illustrated. New York, 1954. Grune & Stratton, Inc. *Price* $16.50.

When we received this volume, our first thought was, "Why review a work intended only for medical physicians in a journal devoted only to surgical problems"? After reading the book it became our opinion that all physicians, no matter what their specialty, could read and reread this excellent work with great profit. Dr. Walter L. Palmer has written the Foreword, dated February, 1954. I can write no better review than to quote from Dr. Palmer's evaluation: "The magnitude of our ignorance of the liver may be illustrated by the fact that it is the only organ in the body whose experimental removal causes death for reasons yet unknown. . . Perhaps it is not surprising, therefore, that to many medical students and practitioners the liver is simply a large organ in the right upper abdomen, physiologically concerned with digestion and clinically troublesome because of the production of gall stones, distension of the capsule in heart failure, enlargement with metastatic neoplasm, and because of ascites or bleeding esophageal varices secondary to cirrhosis—beyond which the liver is a sort of no man's land. . . It is fortunate indeed that Dr. Spellberg has seen fit to assemble so beautifully in this volume such a mass of knowledge of the liver—its structure, function, and derangement in disease. The thoroughness of his work is evident from the fact that nothing seems to have been omitted. The bibliography of approximately 2,400 references testifies to the exhaustiveness of the study. . . The therapy of hepatic disease is amply discussed with clear explanations of basic principles and reasons. . . The brief summaries in bold type at the end of each section will be greatly appreciated by those wishing to find or digest quickly the most important information on any one subject. . . It is a pleasure to congratulate Dr. Spellberg and to express appreciation for this splendid compilation of the existing knowledge of that most mysterious organ, the liver."

We agree with Dr. Palmer. Dr. Spellberg has written a splendid scientific work.

Maxillofacial Anatomy. With Practical Applications. By Harry H. Shapiro, D.M.D. 392 pages with 314 illustrations, 46 in color. Philadelphia, 1954. J. B. Lippincott Co. *Price* $12.00.

After we had carefully read, and in parts reread, this work on maxillofacial anatomy, we turned to the Preface in which the author writes:

"This book is specifically designed to suit the particular needs of students and practitioners of dentistry and medicine for practical information on the morphologic, functional and roentgenologic anatomy of the face, the jaws and the associated structures of the head and the neck. In addition to the description of normal structure, a number of abnormal and

anomalous aspects of the anatomy are included and are discussed in relation to treatment.

"Interest in the study of anatomy is heightened, and the subject matter is better understood and retained when taught in relation to its usefulness in practice. For these reasons, and also because a working knowledge of the anatomy is of prime importance in practice, significant applications are woven into the text, exemplifying the need for a correlated study of the basic structures."

This quoted portion explains the book—a treatise on a special anatomic section. It has been written mainly for students and practitioners of dentistry, but interns, residents and surgeons also can peruse its pages with the assurance that they will add to their sum total of scientific knowledge.

Amputations. By Leon Gillis, M.B.E., M.B., B.CH., D.L.O. 423 pages with 332 figures, some in color. New York, 1954. Grune & Stratton, Inc. *Price* $12.75.

The reviewer believes that this book is a "must," especially for the general surgeon who often is called upon to perform amputations. The trained and skilled limb surgeon who over the years has had experience with thousands of amputations naturally is the man to lead the way and offer the results of his experience to others. This the author has done. He has tried to apply the lessons he and other leaders in this field have learned. He outlines the conditions requiring operation and then discusses when, where and how to operate. The importance of rehabilitation is stressed. This is followed by a consideration of the prosthesis and how the patient can be trained to use it. Until recent times manufacturers of artificial limbs have been supplying their products and recommending the type which in their experience has been found most suitable; and although the surgeon has performed an excellent operation, it has not always been capable of the best prosthetic results. Often reamputation has been in order or the patient has been sentenced to a life of invalidism. There is no reason to outline the scope of this work; suffice it to say the book covers the field in detail. It is beautifully done, well written and illustrated by a master technician and surgeon.

Surgery of the Small and Large Intestine. By Charles W. Mayo, M.D. 340 pages with 94 plates. Chicago, 1955. Year Book Publishers, Inc. *Price* $9.00.

This is the latest of the publishers' series under the imprint "The Handbooks of Operative Surgery." It is written by a surgeon with a great clinical background and experience. The first chapter is devoted to the diet of the patient who is to be operated upon for various intestinal conditions. The postoperative diet in also considered in detail. Specimen diets are given. The technics of the various operative procedures are presented in line drawings on the right hand page. (There are ninety-four plates, each of which contain up to six drawings.) The text description of these various stages in an operation fills the left hand page. Conscientiousness and lack of repetition are striking. This book will prove invaluable to the intern on a surgical service, to surgical residents and to young surgeons. It is a useful book in every way.

Anatomy. Regional and Applied. By R. J. Last, M.B., B.S. (Adel.). 665 pages with 309 illustrations, many in color. Boston, 1954. Little, Brown & Company. *Price* $10.00.

This book on anatomy has a new approach; its aim is the encouragement of the reader or student to understand and appreciate anatomy.

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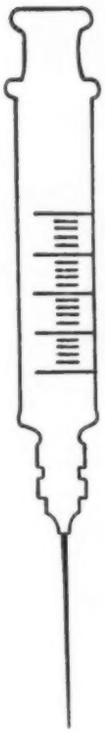

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References: 1. Gershenfeld, L., Am. Jl. Pharm.: 126:112, 1954.
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1. Hingson, R.A., Davis, H.S., Inman, C.E., and LeLievre, R.E.: Am. Pract. 6:1004 (July) 1955

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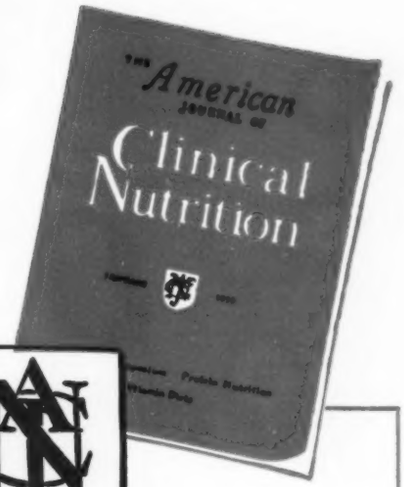

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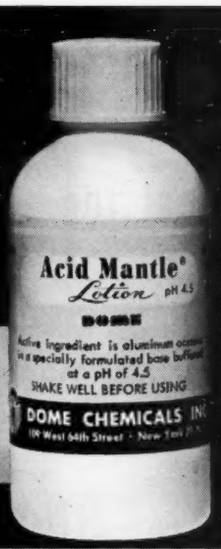

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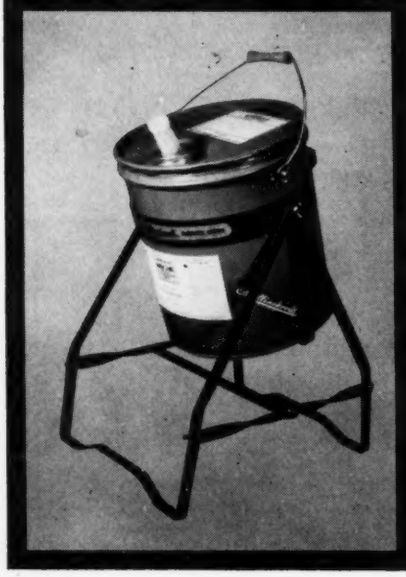

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1. Deuel, H. J., Jr.: Newer Concepts of the Role of Fats and of the Essential Fatty Acids in the Diet, Food Res. *20*:81 (Jan.-Feb.) 1955.

2. Meng, H.C.: Preparation, Utilization, and Importance of Neutral Fat Emulsion in Intravenous Alimentation, in Najjar, V.A.: Fat Metabolism, Baltimore, The Johns Hopkins Press, 1954, pp. 69-92.

The nutritional statements made in this advertisement have been reviewed and found consistent with current medical opinion by the Council on Foods and Nutrition of the American Medical Association.

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References: 1. J.A.M.A. 153:1516, 1953. 2. M. Clin. North America 27:189, 1943. 3. Surgery 18:200, 1945. 4. N.N.R. 1954, p. 107.

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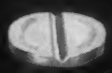
NEW 1 mg. tablet |  5 mg. tablet

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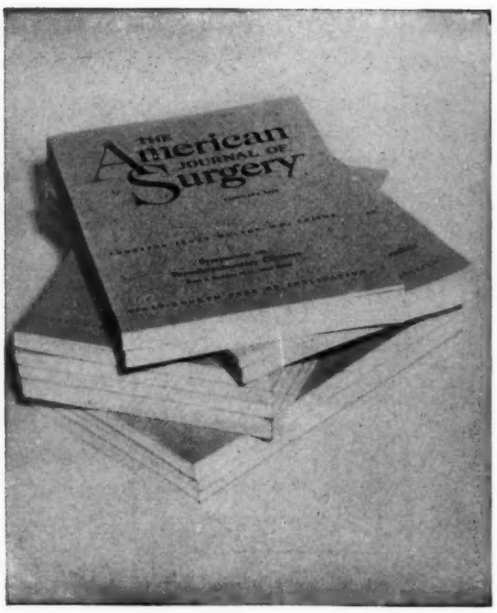

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NOTES AND QUOTES

Two doctors, Harold Lamport, Research Associate in Physiology at the Yale School of Medicine, and Herbert F. Newman, Attending Surgeon at Gouverneur Hospital in New York City, reported recently that they had developed an ultrasound technic in experiments on cadavers for using high frequency waves to break up urinary stones.

The report on their six-year study of various methods for drilling through and disrupting urinary stones appeared in a recent issue of the *Yale Journal of Biology and Medicine*. While some of the drilling methods they describe are promising, the best was the ultrasound drill applied by a technic successful in cadavers but not yet tested in human beings.

This research opens up the possibility that stones which continue to block the urinary passages, even after the best efforts at removal by a urologist, can still be treated without surgery. If such stones can be harmlessly fragmented in patients by the ultrasonic method developed in cadaver experiments, the small pieces of stone will be flushed out or passed in normal urine excretion.

The attention of physician medical writers is called to the Mississippi Valley Medical Society Annual Essay Contest. Any subject of general medical or surgical interest including medical economics and education may be submitted, provided the paper is unpublished and is of interest and applicable value to general practitioners of medicine. Contributions are accepted only from physicians who are members of the American Medical Association and who are residents and citizens of the United States. Manuscripts must not exceed 5,000 words and should be submitted in five complete copies, in manuscript style. The winning essay will receive a cash price of $100.00, *a gold medal and a certificate, as well as an invitation to address the annual meeting of the Mississippi Valley Medical Society, which is held at the same time and place as the annual meeting of the American Medical Writers' Association. (The 1956 meeting will be held at the Hotel Morrison in Chicago on September 26, 27 and 28.) The Society may also award certificates of merit to physicians whose essays rate second and third. Essays must be in the office of the Secretary of the M.V.M.S. not later than May 1, 1956. Winning essays are published each year in the January issue of the* Mississippi Valley Medical Journal. *Further details may be secured from the Mississippi Valley Medical Society, Dr. Harold Swanberg, Secretary, 209–224 W.C.U. Building, Quincy, Ill.*

THE AMERICAN SOCIETY OF MAXILLOFACIAL SURGEONS announces an Essay Contest for papers based on Clinical and Experimental Research in Maxillofacial Surgery.

A first prize of $350.00 and a second prize of $200.00 is offered for suitable Essays. Any medical graduate is eligible to enter a paper, which must be between 2,000 and 4,000 words in length, and based on original work not previously published.

Closing date for submission of Essays is March 1, 1956. Further information can be obtained from the Secretary of the Society, Dr. John A. Drummond, 1414 Drummond Street, Montreal 25, Canada. The winning papers are to be read before the Annual Meeting of the American Society of Maxillofacial Surgeons to be held at the Jung Hotel, New Orleans, Louisiana, March 19 to 21, 1956.

Veterans Administration announced it will add another area medical office to the six now in operation so as to provide more efficient supervision of medical activities and thereby give more effective service to veterans. Columbus, Ohio, has been selected as the site for the new area medical office, but the location and the opening date will be announced later.

Dr. John G. Hood, present manager of the V. A. Hospital in the Bronx, New York City, has been appointed Director of the new office. Dr. Hood has been with V. A. since 1930. In addition to his present position as manager of the Bronx hospital, he has served as manager of V. A. hospitals in Richmond, Virginia, Atlanta, Georgia, and Perry Point, Maryland.

AN exhibit of thirty original oil paintings in the unique series, "A History of Pharmacy In Pictures," was recently held at the Smithsonian Institution. The showing was the first formal public display of the full color paintings

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NOTES AND QUOTES

in the East. Coincident with National Pharmacy Week, the exhibit was hosted on the opening day by the man who conceived and developed the series, George A. Bender of Parke, Davis & Company, and the artist, Robert A. Thom.

The paintings were commissioned in 1951 by Parke, Davis as a tribute to the traditions of the pharmaceutical profession. They show dramatic highlights in the field of pharmacy from "Before The Dawn of History" to "A Revolution in Pharmaceutical Education." This latter painting shows a University of Michigan classroom in about 1871, with Dr. Albert B. Prescott, pioneer in pharmaceutical education, addressing his class.

Of interest locally is the twenty-seventh painting in the group which depicts the founding in 1852 of the American Pharmaceutical Association, now headquartered in Washington. An explanatory caption says that because of the need for intercommunication, educational and apprenticeship standards and quality control of imported drugs, a convention of representative pharmacists was called to meeting in the Hall of the Philadelphia College of Pharmacy, just 103 years ago.

The University of Texas M. D. Anderson Hospital and Tumor Institute announces that facilities and equipment for therapy and research in the field of malignant diseases are now provided for qualified applicants seeking training and research opportunities in oncology: 80,000 *feet of space devoted to research, Clinics equipped to handle* 100 *new patients and* 200 *revisits daily, a Cobalt-60 Irradiator and Betatron, and radioactive isotope facilities. Fellowships and residencies are under the auspices of The University of Texas Postgraduate School of Medicine. Participation in fundamental cancer research under able guidance, as well as a broad program of education in the Texas Medical Center, is offered in the following divisions: Diagnostic Radiology, Therapeutic Radiology, Anatomic Pathology, Clinical Pathology, Experimental Pathology, Medicine, Experimental Medicine, Surgery, Experimental Surgery, Malignant Diseases (Rotating), Anesthesiology, Biochemistry, Pediatrics, Gynecology, Physics, and Neurologic Surgery. For further information and application forms write to: Dr. Grant Taylor, Office of Education, The University of Texas M. D. Anderson Hospital and Tumor Institute, Texas Medical Center, Houston 25, Texas.*

For the first time in medical history Veterans Administration will make an extensive evaluation of mental patient care to determine what types of treatment and hospitals best promote the improvement or recovery of its psychiatric patients and the relative costs.

V. A., which has one of the nation's largest psychiatric patient loads under a single medical management, said the preliminary phases of the pioneering project have been started.

The study will continue for five years, with probably fifteen of V. A.'s forty neuropsychiatric hospitals participating, according to Dr. Jesse F. Casey, Director of Psychiatry and Neurology Service.

Dr. Casey said the information to be sought is not now available from any source. When it is completed, he said, it should go a long way toward establishing psychiatric care on a much higher level of effectiveness and thereby speed the recovery or improvement of psychiatric patients at the least possible cost.

The average American today owes the last twenty-one years of his life to medical science, and even greater achievements are in store if medical education is supported, the hospital, surgical and optical industry has been informed.

The nation's dependence on the teaching and research programs of the eighty-one medical schools was outlined in detail in a new 6,000 *word analysis of the crisis in medical education made available by Mr. Charles H. Prange, President, Austenal Laboratories, Inc., and Chairman of the Hospital, Surgical and Optical Division of the National Fund for Medical Education.*

The study, entitled "Handbook for Medical Education," points out that the schools need $10,000,000 *additional annual income if they are to meet constantly mounting demands for more doctors and more research. Whether this country reaps the full medical benefits of atomic research may depend in part on corporation support of the medical schools.*

CONTINUED ON PAGE 50

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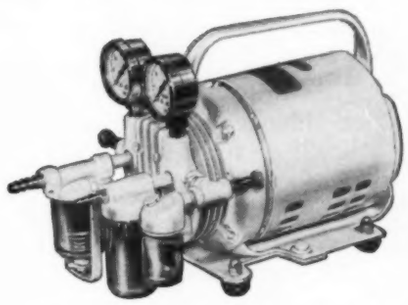

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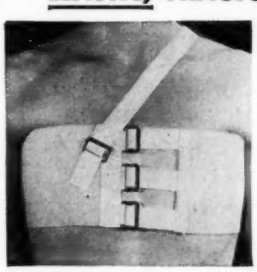

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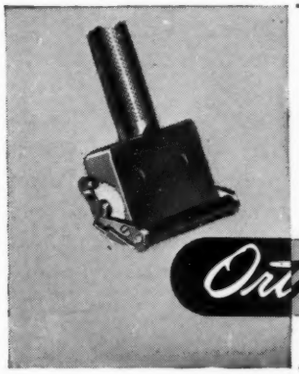

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NOTES AND QUOTES

PEOPLE in the farm states of the midwest live longer than those in other areas of the United States, according to the Metropolitan Life Insurance Company's statisticians.

Expectation of life at birth in the West North Central area at the mid-century was 67.8 years for white men and 73.3 years for white women. The next best record for men is found in New England—66.9 years, and for women on the Pacific Coast—72.9 years.

Average length of life increased substantially in every section of the country during the decade of the 1940's, according to life tables computed by the statisticians from data provided by the National Office of Vital Statistics and the Bureau of the Census.

The largest gains were made in the areas which formerly had the least favorable records. For example, in the Mountain States the increases were 4.4 years for white men and 5.9 years for white women. This compares with increases of 2.6 and 4.1 years, respectively, in the West North Central region.

Dr. William H. Masters said that he was completely convinced that a third sex, or "neutral gender," does exist and that the members are former males and females who have reached approximately the age of sixty. Some women reach the neutral gender age before fifty, others not until seventy. He believed it was safe to generalize that the female was roughly fifteen years ahead of the male.

This report came from Dr. Masters as he presented a paper on "Problems of Endocrine Therapy in the Ageing Individual," before 1,200 specialists of The American Academy of Obstetrics and Gynecology at The Fourth Annual Clinical Meeting of The Academy which was held in Chicago last December.

THE newest technics of heart surgery make use of circulation outside the body and give the surgeon one-half hour to work on a bloodless heart.

Dr. William P. Young of the University of Wisconsin Medical School told doctors attending a postgraduate course on heart diseases of this new technic. Arterial blood is pumped from a bottled blood reservoir into the large subclavian artery. The blood circulates normally through the body, but before it enters the heart through the vena cava, it is shunted out of the body to a bottled venous reservoir. Arterial blood is supplied constantly, and venous blood removed from the body without passing through the heart.

All body tissues receive a blood supply, but the heart itself is bloodless, permitting surgery.

Personality disturbances in patients with tuberculosis do not follow a consistent pattern, as with peptic ulcers or ulcerative colitis, but some reactions to the disease suggest a psychologic hibernization, according to Dr. Jules V. Coleman, clinical professor of psychiatry, Yale University of Medicine, writing in a recent issue of the Bulletin of the National Tuberculosis Association.

In general, Dr. Coleman says, these reactions are associated with feelings of helplessness, of being unable to cope with the problems at hand by means of accustomed routines of living.

"This common response may be considered a kind of psychological hibernization, i.e., it is as if the patient winterizes out his disease by withdrawing into a protective covering which excludes or minimizes external stimuli and reduces psychological response to external demands to a very much lower level," states Dr. Coleman. "The degree of psychological withdrawal depends largely upon the particular patient's personality and may at times be very extensive."

Dr. Coleman points out that physicians are seeing more patients in general whose complaints are almost entirely emotional in origin. This is presumably related to better control of communicable disease and to economic factors making medical care accessible to more people than formerly, he states. Sometimes, he continues, physicians treat their patients' emotional conditions with placebos instead of attention to the emotional problems.

"The treatment of disease," he writes, "would be a much simpler matter if only reasonable people became ill, or if people remained reasonable after becoming ill. However, irrational reactions to illness are hardly the exception, and must be themselves regarded as symptoms, especially of prolonged or chronic diseases. In other words, an illness is always a psychological as well as a physiological disorder."

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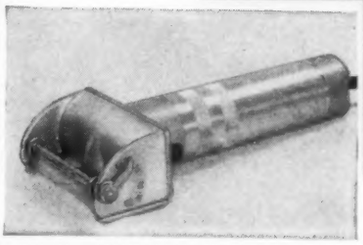

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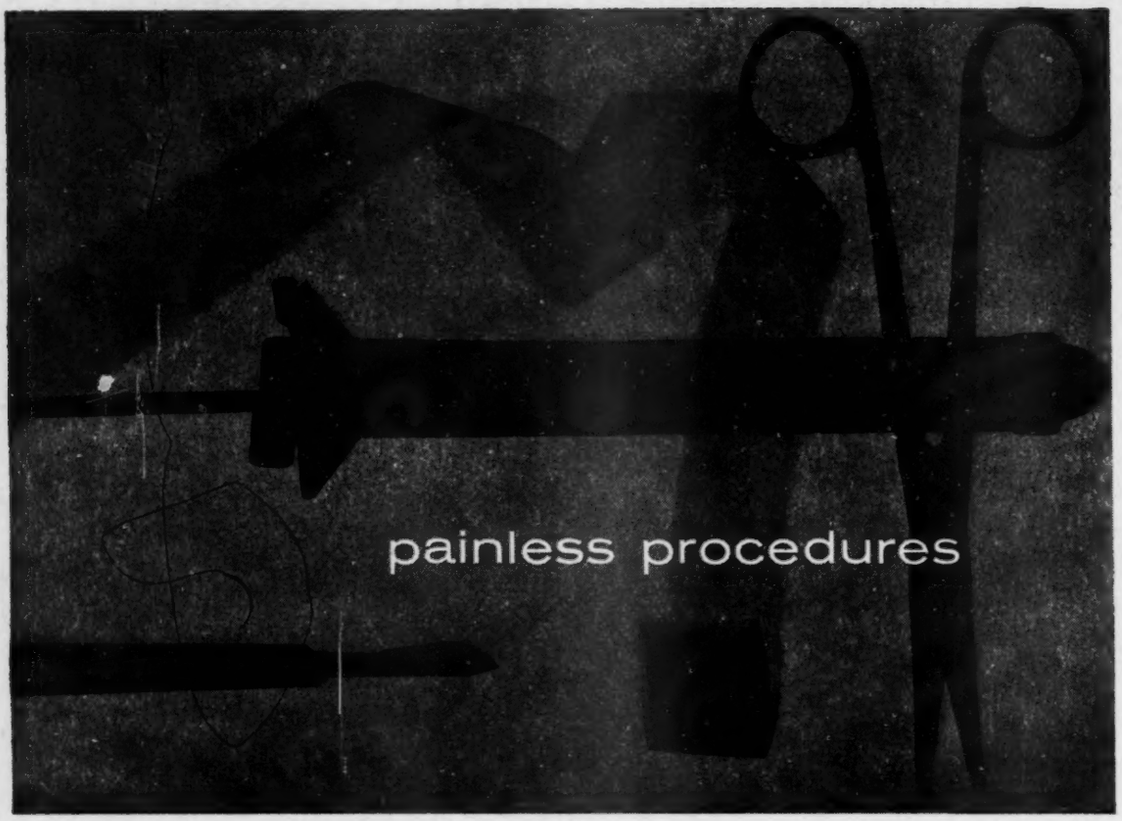

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